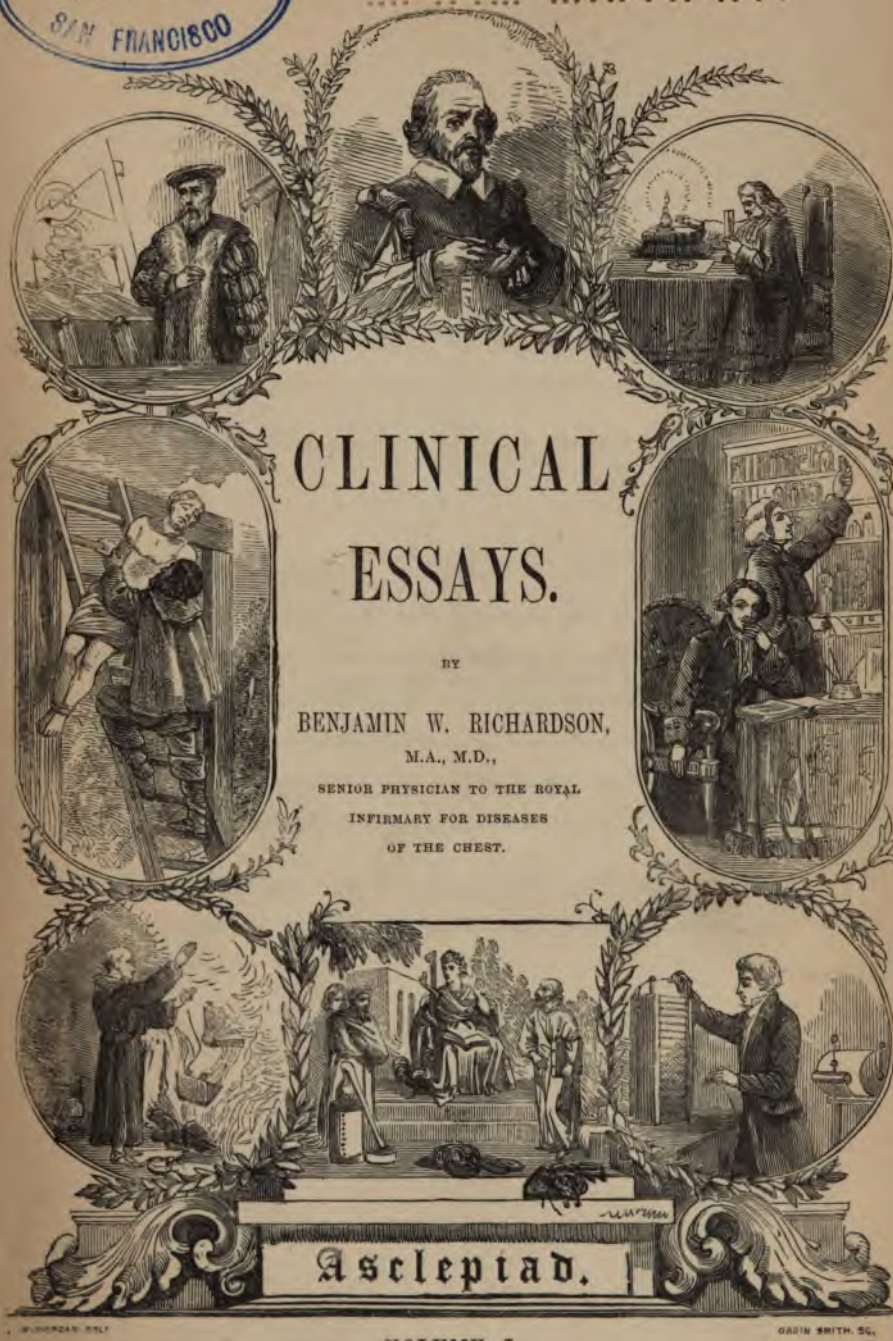

CLINICAL ESSAYS.

BY

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TO

J. A. EASTON, M.D.,

FELLOW OF THE FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW;
AND
PROFESSOR OF MATERIA MEDICA IN THE UNIVERSITY OF GLASGOW.

MY DEAR PROFESSOR,

I always recall with gratitude and pleasure the fact that in your *Asclepia* I first read the tablets of medicine; and have long courted the opportunity of expressing publicly how much I am indebted to your profound learning, your experience, and your kindness, which, communicated at first to the student, have been extended unremittingly to the practitioner and author.

That you may long live, and long fill the position you have so deservedly earned in medical science, is, my dear Professor, the sincere wish of

Your affectionate and obliged friend,

BENJAMIN W. RICHARDSON.

PREFACE.

In the Asclepiad it is my intention to bring out in occasional volumes, after the manner of the volume now presented, a series of essays on subjects relating to medical science in its various departments. I cannot promise in this series any distinct regularity of publication, inasmuch as time, labour, and thought are not altogether at my disposal; but I hope that the volumes will, on an average, be issued half-yearly, or, at least, annually.

While it will be my object in this series always to consult, as far as in me lies, the requirements of practical medicine, I shall endeavour to weave into the subjects discussed such matters bearing on the current theories of medicine as shall tend to open the way to new and more comprehensive views, and to a sounder and more rational practice.

I wish it to be understood that if, in progress of time, any expression or opinion which may be

advanced should prove on reasonable evidence to be incorrect, I shall not fail to make the correction. Nor shall I fear the charge that one volume is not strictly in accord with its predecessor; for, as Emerson aptly remarks, "unyielding consistency is the hobgoblin of weak minds;" and, as in medicine many things are still very obscure, and all things admit of being re-learned and re-taught by the light of advancing knowledge, so it is not for the student to dogmatise and to refuse to re-construct because he has previously constructed.

12, Hinde Street, Manchester Square,
November 28th, 1861.

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CLINICAL ESSAYS.

ESSAY I.

ON SUBCLAVIAN MURMUR.

In conducting auscultatory examinations of the chest, the physician now and then meets with a passing murmur under one or both clavicles. To the inexperienced, it is difficult to determine whether the sound is connected with respiration or with the circulation; for it obtains in many cases that the sound, which seems arterial, is so peculiarly associated with the respiratory act, that its source assumes a double character. The sound has been described by few authors, but it is known to most persons who are extensively engaged in stethoscopic inquiries. I once heard Dr. Kirkes read a concise paper on the subject; Dr. Sibson has also referred to the murmur in explanation of its origin; and Dr. Thorburn, in the *British Medical Journal* for June 18th, 1859, has entered into a description of it in a paper entitled "A Peculiar Auscultatory Phenomenon". Dr. Thorburn, in certain doubts which he offers as to the origin of the

sound, expresses what is, I believe, a general feeling. I shall endeavour in the succeeding observations to clear up the obscurities of the phenomenon, and to exhibit it in its true and simple light. The importance of the labour, in a diagnostic sense, I cannot better explain than by stating the fact that, within the last three years, four cases have been referred to me as cases of aneurism in the thorax, which have proved on further examination to be nothing more than well marked examples of subclavian murmur; cases, as we shall hereafter see, very innocent in their nature when compared with aneurismal disease.

I will follow out the description of subclavian murmur from its history, as it is written in the natural facts.

OBSERVED FACTS AND ANALYSES.

In 1856, a young man came before me at the Royal Infirmary for Diseases of the Chest, complaining of great lassitude, thirst, flatulency, depression of spirits, and palpitation of the heart. He was a tall, thin, anæmic man, by trade a cabinet-maker, and evidently of an irritable and what is popularly called "nervous" temperament. On examining his chest, I was surprised to hear under each clavicle a sharp loud murmur, synchronous with the systole of the heart, and resembling closely an aneurismal *bruit*. Tracing the murmur downwards towards the heart, I found it becoming reduced; whilst over the heart it was absent altogether. Each cardiac sound was clear, well defined, and natural. I followed out the courses of

the carotid, axillary, and femoral arteries with the stethoscope, and found in them no murmur. Percussing the chest in the subclavian regions, I discovered perfect resonance; but, on placing my fingers over the subclavian artery on each side, there was a distinct fremitus with each pulsation of the vessel. The patient himself was conscious of this fremitus, describing it as a gentle thrill beneath the fingers. On returning to the stethoscope, and divesting the ear of the murmur, the pure respiratory sounds were audible at every point; but now a further fact came out, viz., that whenever a long inspiration was made and the breath was retained, or whenever a long expiration was made, then the specific arterial sound was lost. I brought this patient to many medical friends, who expressed various opinions as to the nature of the disease; ultimately, however, the evidence became conclusive that the murmur was produced by posterior pressure on the subclavian artery.

The case being for a long time under my care, I made many observations upon it. Amongst these, it was distinguishable that the murmur underwent modification, according to the position of the arm corresponding to the side examined. When the arm was brought down close to the body, the murmur was lost; as the limb was raised towards a right angle, the sound recurred; at a perfect right angle, it rose to its acme; but at an acute angle, made by raising the limb towards the head, the sound declined; and when the limb was quite vertical, it was nearly lost.

From the time I have named, until the present year, the patient was under observation. He recovered his health on the administration of steel and quinine, and followed his business until 1859; but the murmur was always present, being specially developed after any excitement, as after a sharp walk, or mental worry. The last time I saw this man, he was suffering from distinct indications of tubercular deposit in the apex of each lung. He left London for the country; and has returned, I am informed, so much improved, that he is able to follow his work.

The occurrence of this case led me carefully to investigate other patients for the same symptom. I soon found that the murmur was an accompaniment of certain special diseased conditions, more or less severe. Thus in 2,000 patients of all ages, 1,011 of whom were females, and 989 males, who came under my care at the Royal Infirmary for Diseases of the Chest, from the 11th of November, 1858, to the 17th of August, 1860, and each of whom underwent careful examination for the murmur, I found it well marked in 51 cases, *i. e.* in 2·55 per cent. Among the 51, the murmur was found in enormous preponderance in males; there being 48 of the male sex presenting it, and 3 only of the female. The youngest patient in whom the murmur was heard was one John Clark, a coppersmith by trade, eighteen years old; he was suffering from simple dyspepsia: the oldest patient was one Edward Butler, a carpenter, seventy-four years old; he was labouring under subacute bronchitis.

The 2,000 patients to whom reference has been made, came with various diseases; there were cases of phthisis, of chronic bronchitis, of anæmia, chronic pleurisy, different forms of heart-disease, thoracic aneurism, stricture of the œsophagus, dyspepsia, diabetes, empyema, spasmodic asthma, morbus Brightii, cirrhosis of the liver, and a long list more. But the murmur itself was met with in five diseased states only; viz., in chronic bronchitis; in phthisis (during the first and second stages); in anæmia; in dyspepsia; and in valvular disease of the heart.

The cases of *bronchitis*, chronic and subacute, in the total of 2,000, were 610 in number, males and females together. Subclavian murmur was well marked in 18 of these; viz., in 16 males, and in 2 females. The bronchial affection was generally of long standing, and the mischief wide spread. The marked signs of disease were not always in the lung of the side on which the murmur was most distinct. There were three cases of subacute bronchitis.

The total cases of *phthisis* subjected to examination were 669. Of this number, 431 were males, and 238 were females. The murmur was presented in 12 of these patients, all of them being males. The tubercular disease, at the period when the murmur was well marked, was situated in the apex of the lung on one or both sides; the murmur was most distinct in the early stages of tuberculosis.

There was one mixed case, in a man aged 23, in whom, with old standing bronchial disease, there was evidence of tubercular deposit in the apex of the

right lung. In this example the murmur in the subclavian region was well marked, but only on a slightly forced inspiration.

The cases of *anæmia* were 172, males and females together. Out of these, there were 10 examples of subclavian murmur; viz., in 9 men, and in one woman. They were all patients who, shut off from fresh air, had been also overworked and badly fed; their symptoms were the common ones of anæmia, without any apparent organic lesion: they all recovered under steel and an improved hygiene. In two cases there was anæmic murmur in the neck, which subsided on recovery. In these cases, as the general health improved, there was less intensity of subclavian bruit, but it did not disappear as a sound.

The total of the cases of *dyspepsia* was 281, males and females. In these, the murmur was present in seven instances, all men. The dyspeptic symptoms were those of pain after food, irregular appetite, eructations, and flatulency. The cases were not serious, and they recovered under an alterative tonic treatment with revised dietary and habit; but in every case the murmur remained after the recovery seemed complete.

The examples of *disease of the heart* were 93 in number out of the 2,000; they were mainly chronic cases. The subclavian murmur was present in three of these patients, all males. Two cases gave the stethoscopic signs of mitral, the other of aortic valvular disease. They improved under tonic treatment, and

were discharged relieved. The subclavian murmur, however, continued.

I would remark in this place on the singular fact that, in the list of diseases in which subclavian murmur was an attendant symptom, there is not one example of aneurism ; yet there were included, in the 2,000 cases, no fewer than six instances of aneurism, either of the aorta itself, or of the innominata, or of both. In these, aneurismal *bruit* was most evident, but without any special extension into the subclavian on either side. There was also one case, in which I diagnosed atheroma of the aorta, and in which a loud murmur extended along the whole line of the aortic arch, and upwards towards the neck. After death, I found atheromatous disease of the arch, extending into the trunks of the three great vessels springing from it. Nevertheless, this case supplied no evidence during life of the specific subclavian murmur at the present time under discussion.

To illustrate further the 51 cases of subclavian murmur, I have entered them down in the following table, with particulars relating to age, sex, disease, duration of disease, and occupation. The ages and other facts refer to the time when each patient first came under my observation. The occupations are classified together when two or more patients followed the same: the reason for this will be hereafter shewn.

No. of Case.	Patient.	Age.	Disease.	Duration of Illness.	Occupation of Patient.
1	Edward Wade	30	Anæmia: ultimately phthisis.	1 year.	Cabinet-maker.
2	John Creep	24	Anæmia.	2 years.	Ditto.
3	Edward Ellis	40	Phthisis pulmonalis.	3 months.	Ditto.
4	Benj. Johnson	36	Mitral disease of the heart, and great debility.	Acute rheumatism 2 mo. before admission. Had been well to that period.	Ditto.
5	Wm. Blandford	51	Chronic bronchitis.	18 years.	Ditto.
6	Robert Brown	32	Chronic bronchitis.	6 weeks.	Ditto.
7	John Bolton	44	Chronic bronchitis.	4 years.	Ditto.
8	Edward Collis	40	Phthisis pulmonalis.	6 months.	Ditto.
9	George Stretcher	18	Phthisis pulmonalis.	Sickly 12 months.	Ditto.
10	James Gill	49	Subacute bronchitis.	4 weeks.	Ditto.
11	Wm. Grainger	24	Phthisis pulmonalis.	18 months.	Ditto.
12	Geo. Thatcher	18	Phthisis pulmonalis.	15 months.	Ditto.
13	James Slater	29	Anæmia.	8 months.	Ditto.
14	Edward Brown	48	Chronic bronchitis.	15 years.	Ditto.
15	Richard Fuller	69	Subacute bronchitis.	1 month.	Ditto.
16	George Moody	21	Anæmia.	2 months.	Ditto.
17	Richard Bull	40	Chronic bronchitis.	4 months.	Ditto.
18	William Ambler	26	Phthisis pulmonalis.	4 months.	Carpenter.
19	Edward Butler	74	Subacute bronchitis.	1 month.	Ditto.
20	Thomas Ford	38	Chronic bronchitis.	2 years.	Ditto.
21	Richard Hebber	64	Chronic bronchitis.	1 year.	Ditto.
22	Thomas Gray	40	Chronic bronchitis.	2 years.	Ditto.

23	George Pond	23	Phthisis pulmonalis.	5 months.	Box-maker.
24	Job Hopkins	42	Chronic bronchitis.	6 months.	Sawyer.
25	John Tatum	22	Dyspepsia.	6 months.	Ditto.
26	William Manby	23	Phthisis pulmonalis.	3 months.	Baker.
27	Joseph Herring	60	Dyspepsia.	1 year.	Ditto.
28	Samuel Crocker	34	Subacute bronchitis.	3 weeks.	Clock-maker.
29	Charles Topley	30	Dyspepsia.	3 weeks.	Ditto.
30	Denis Ryan	22	Phthisis pulmonalis.	Sickly 10 years.	Shoemaker.
31	Fred. Webster	28	Aortic valvular disease.	8 months.	Ditto.
32	John Clark	18	Dyspepsia.	15 months.	Coppersmith.
33	Thomas Budden	30	Phthisis pulmonalis.	2 years.	Gunsmith.
34	George Speer	24	Anæmia.	8 months.	Vellum-binder.
35	William Castro	18	Anæmia and palpitation.	Palpitation 1 week.	Watch case engine maker.
36	Fran. Woodgate	43	Anæmia.	6 years.	Gold-beater.
37	John Thompson	36	Acute dyspepsia.	1 week.	Cab-driver.
38	Uriah Wilmot	30	Dyspepsia.	8 months.	Vocalist.
39	George Custance	37	Subacute bronchitis.	3 weeks.	Cigar-maker.
40	John Edwards	26	Anæmia.	2 years.	Brass finisher.
41	Francis Franks	33	Chronic bronchitis.	2 months.	Hemp-dresser.
42	Fred. Arnold	26	Mitral disease of the heart.	4 months ?	French polisher.
43	Thomas Propert	34	Phthisis pulmonalis.	10 months.	Stone-mason.
44	George Business	29	Acute phthisis.	3 weeks.	Artificial flower maker.
45	John Francis	32	Dyspepsia.	3 months.	Tool-cutter.
46	Chas. Macnelly	23	Chronic bronchitis and phthisis.	Bronchial cough 3 yrs.	Printer.
47	Jonathan Soden	36	Anæmia.	5 weeks.	Pianoforte-maker.
48	John Huggett	19	Chronic bronchitis.	2 years.	Upholsterer.
49	Brunella Brooks	24	Anæmia.	3 months.	Domestic servant.
50	Jane Halsey	40	Chronic bronchitis.	3 years.	Housewife.
51	Mary Sutton	43	Chronic bronchitis.	3 months.	Ditto.

From this table, we learn certain laws relating to the occurrence of subclavian murmur in diseased states, which are of considerable interest. A few illustrations may be given.

In the first place, there is no case of a child presenting the murmur. The youngest subjects named were eighteen years old. There were, nevertheless, many children included in the total of cases, the majority of whom were suffering from the diseases named in the table. In the case of every child, I examined with more than ordinary care for the murmur. I often found the common anæmic continuous murmur in the neck, but never the subclavian.

Next, we see that the subclavian murmur is rare in females. In the total of cases (2000) from which the deductions were drawn, there were more females than males in the proportion of about one per cent.; yet there were 3 cases only of the murmur on the female side to 48 on the male side. The murmur in the female, moreover, when it did occur, was not to be heard without an effort on the part of the patient to bring it out, such as a long inspiration: it was also much more faint, and even more fleeting, in the women than in the men.

Thirdly, we see from the table, that the murmur as occurring in disease is most peculiar in certain forms of disease. Chronic bronchial affections are those which furnish most illustrations of it, according to my analysis, viz., 18 to the 2000; afterwards stands phthisis, yielding 12 cases; next anæmia, giving 10 cases; next dyspepsia, yielding 7 cases; then heart-disease,

giving 3 cases; and, lastly, a single case of bronchial affection combined with tubercular deposit. These relationships will have to be further considered in the sequel.

Finally, the table shows a curious circumstance in reference to occupation. It will be seen that, in the vast majority of the cases, the patients were employed in manual labours, in the exercise of which the arms are thrown forwards and downwards. Thus there are no fewer than fifteen cabinet-makers in the list; men who are engaged all their lives in planing, sawing, and polishing. There are four carpenters, two sawyers; and so on. When I first observed this connection of the murmur with certain mechanical arts, I began to inquire into the matter carefully. I thought it might be that the men of these trades were more fully represented in the list of patients than others. This turned out to be a fallacy; for, of the 2000, there were many more tailors than cabinet-workers, and more clerks than tailors, and more sempstresses than clerks. I began consequently to inquire how far the special occupation influenced the result. To get at the truth in this direction, I collected eight cabinet-makers who were quite well, and to my surprise found that they all presented the murmur. I extended the observation to carpenters also, and found the same thing, though not so frequently. Blending, therefore, these facts with those which tell us how rare the murmur is in women, and that it is absent in children, I am driven to the inference that particular mechanical actions of the arms

long continued, have an influence in producing the peculiar sound; how and why, we shall see better in a succeeding page.

CAUSE AND CAUSATION OF SUBCLAVIAN MURMUR.

The whole tenor of my observations leads me to the conviction that subclavian murmur is of arterial origin. In this view I shall be supported by the majority of auscultators, but shall be at variance with at least one, I mean Dr. Thorburn, who, speaking of the sound, remarks: "It must either be a respiratory sound modified by the heart and great blood-vessels, so as to have a cardiac rhythm; or a vascular sound modified by respiration. From a careful examination, I have come to the conclusion that it is of the former character: and that what is heard is the expiratory murmur and the end of the inspiratory murmur itself, *saccadée* or jerked by some undue impulse. I think it probable that this impulse is communicated by a nervously excited or perhaps slightly dilated aorta, just as the action of the heart may sometimes give a cardiac rhythm to a friction-sound which is really pleural. The greater comparative weakness of expiration will account for its being heard chiefly at that time."

I have said that Dr. Thorburn is alone in his opinion; but his hypothesis is ingenious, and well put. It is necessary, therefore, in disposing of it, to use careful and logical argument in favour of the opposite view; viz., *that subclavian murmur is an arterial sound, modified by respiration.* The proofs of

this are demonstrative. The murmur is synchronous with the systole of the heart, and with the radial pulse. It is also limited to the traject of the subclavian artery. Traced downwards towards the heart, it is lost; traced in any other direction over the chest-surface, it is lost. Moreover, it is often limited to a certain point in the course of the artery, the space most common to it being a horizontal line about an inch long beneath the middle of the clavicle, or verging a little towards the outer end of the bone. A respiratory sound would, of course, have no such limitation. It happens truly, in many examples, that some modification of respiratory movement occurs antecedently to the murmur. Thus, I have met with many instances in which the murmur was present at no other time than during a deep and sustained inspiration; whilst in other examples, where it has been present during an ordinary inspiration, it has been *destroyed* by deep and sustained inspiration; but as, in numerous cases, it also happens that, by changing the position of the arm, the murmur may be intensified, or lessened, or destroyed, independently of the respiration, it follows that the murmur is not respiratory. The character of the sound, again, is different both from a respiratory sound and a friction sound; it is essentially a pulsatile *bruit*, in which respect it approaches very nearly to an aneurismal murmur.

The view of the arterial origin of subclavian murmur is further corroborated by examples where the thrill of the vessel can be felt by the finger at each

pulsation. In one instance, where this thrill was intense, I could see the vibration when the finger was removed; and not only so, but on applying the ear near to the artery, without actual contact with the skin, I could hear the murmur.

Lastly, it is not difficult, in applying the stethoscope over the subclavian, to catch the murmur by a light pressure, and to remove it by a firm pressure, of the mouth of the tube. In such cases, the stethoscope, by arresting the current of blood through the artery when the pressure is made considerable, removes the sound by stopping the circulation. Under such pressure, the radial pulse is also felt to be deficient or absent.

The cause of the vibration of the artery is nevertheless primarily external; that is to say, the sound is in the artery, but is not due to disease of the vessel. I imagined at one time that in some cases, where the murmur was very intense, there might be deposit on the inner surface of the artery; but this idea was soon dispelled by the observation, that in every case the murmur could be removed by some simple change in the position of the arms of the patient, or in the force of respiration.

The view advanced by Dr. Kirkes as to the cause of the murmur was to the effect that the artery, at the moment when the sound was produced, was subjected to pressure upwards and forwards by a portion of solidified lung raised during inspiration. This explanation, which is the one also given by Dr. Sibson, in so far as it assigns to external pressure upon the

artery the first step towards the production of the murmur, must be taken, I assume, as correct; it is a view to which I was led very early in my inquiries, and it accords with the facts. At the same time, it must not be conceived that the external pressure brought to bear upon the artery to produce the murmur is invariably exerted by the lung; such a view would not accord with *all* the facts.

There are, I believe, two modes by which the pressure on the artery may be exerted, when the subclavian murmur is present.

Firstly, there is a class of cases in which the pressure is produced *by diseased lung*. In these examples, there is, I believe, mostly, some solidification at the apex of the lung on the side on which the murmur is heard; the solidification may be due to deposit of tubercular matter, or to enlarged and indurated bronchial tubes. In such instances, the character of the murmur varies according to the condition of the lung-substance. If there is a diffused tubercular matter, with general dulness on percussion and deficient respiration, the murmur is only to be elicited at the acme of a deep inspiration. It is then heard very softly, as a gentle fleeting coo, irregular in its occurrence, and often not distinguishable without difficulty from a reflected heart-sound.

In other cases, where tubercular deposit is laid down in one spot immediately in contact with the artery, and where the deposit is hard, or when there is enlargement and induration of bronchial tubes immediately behind the artery, the murmur, which

will still only be heard during inspiration, is sharp and shrill, or shrill and grating. It will continue so long as the breath is being held, and will disappear immediately on expiration. The murmur thus caused is often well marked in the early stages of phthisis, but disappears in later stages, when the deposit of tubercle has undergone the changes of softening and removal.

In another class of cases, where chronic bronchial disease is at a distance from the subclavian region, the pressure seems to be made on the artery by healthy lung acting under undue distension for compensation. In these examples, the murmur is very soft and fleeting, occurring only at the acme of inspiration.

Secondly, the murmur may be presented *without any disease in the structure of the lungs.* It will be seen, indeed, on reference to the table already given, that out of the fifty-one cases I observed, there were no fewer than nineteen in which pulmonic disease was absent; added to which, I have seen many examples of the murmur in persons in perfect health. In all these examples, the pressure exerted on the artery is, in my opinion, brought about by the action of the subclavius muscle, and is the result of those occupations in which the arms are being constantly thrown forwards and downwards, as occurs in wood-planing, hand-sawing, French polishing, and the like.

As I indicated in describing the first case in this essay, position of the arm makes a decided modification

in the sound; that is to say, when the arm is parallel with the body, the murmur is at its minimum, or is absent: when the arm is at a right angle with the body, or a little above the right angle, the murmur is at its acme. This is explained by the fact, that in the first named position the subclavius is relaxed; in the last the subclavius is exerting its contraction to help to steady the shoulder; thus the artery, pressed down by the muscle, is borne towards the margin of the first rib, and the murmur is elicited. This explanation of a modification of murmur by position of the limb bears on the influence of occupation. For, when the arm is thrown forwards, as in planing, and is brought back again by a brisk effort, the subclavius at each movement is brought into active play, the artery is pressed so as to impinge on the rib, and, as this proceeding is repeated for many years, the parts so adapt themselves that the position of the vessel is modified by the circumstances, and subclavian murmur becomes a permanent, but of itself a harmless phenomenon.

The two methods by which the sound is educed are then analogous in their action, but they are very different in their meaning in a prognostic sense. They may be singly at work; or, as will be readily seen, they may be working in combination, as when a man in whom the murmur is present as a mere mechanical effect of muscular contraction, becomes the subject of tubercular deposit or bronchial disease.

These modes of production are sufficient to account for the existence of subclavian murmur, independently,

as we have already seen, of roughness or deposit on the inner surface of the vessel. It would appear, indeed, from the labours of Chauveau and Marey, that roughness of the internal surface of arteries does not anywhere, nor in any case, give rise to murmur. But the question is still open for discussion, whether the condition of the blood produces this modification. Agreeing with the opinion that arterial murmur is due to contraction of the artery at the point of murmur, and to the vibration incidental to a change in the arterial tension, I cannot but state that in many cases where I have met with *intense* murmur, there have been signs of anæmia; and that the murmur has borne a relationship in intensity with the more determinate indications of the anæmic condition. There are various methods of explaining this observation, the best of which is that of Marey; viz., that in anæmia there is a feebler arterial tension and less resistance to the current of blood in the capillaries. But whatever the explanation, it is certain that the relationships I have pointed out are true; and that anæmia, though not a cause of the murmur, has a qualifying influence upon it.

EXACT CHARACTER OF SUBCLAVIAN MURMUR.

The murmur differs in character in different cases, and sometimes in the same case. It may be a soft musical coo, so short and so slight as to be mixed with and obscured by the respiratory sounds. It may be a loud, coarse, almost snorting gush, conveying an unpleasant thrill to the ear, and, during its transit,

obscuring the vesicular murmur of respiration. It may be loud, and yet so short, as to resemble a sound produced by a quick blow, with dull resonance. These are the three typical forms of the murmur, but between them there may be various distinctive shades. The musical cooing murmur, for example, may be intensified to a whistle, or a ringing noise; and the coarse murmur, to an absolute rasp. The murmur under one or other type is always best developed when it occurs in the right subclavian space, but it is most common on the left side.

DIAGNOSIS OF SUBCLAVIAN MURMUR, DIFFERENTIAL AND ABSOLUTE.

Differential Diagnosis. There are four physical signs of disease, diagnosable by the stethoscope, with which subclavian murmur may possibly be confounded, and especially by those who are not familiar with the details, as well as the principles, of physical diagnosis. These physical signs are—

ANEURISMAL MURMUR.
VALVULAR MURMUR—MITRAL AND AORTIC.
PLEURITIC FRICTION.
BRONCHIAL COO.

Aneurism of the aorta at the arch, or of the innominate artery, or of the subclavian itself, are the first diseases to be differentiated. I have said, that on four occasions I have known subclavian murmur set down as aneurismal; I may add to this, that in one of these cases the life of the patient was refused

for insurance on the suspicion of subclavian aneurism. The following diagnostic points are, however, always sufficient to establish the difference between subclavian murmur and any thoracic aneurism.

In aneurism the character of the bruit is usually the same at all times, the point of maximum intensity being one fixed spot, on which the mouth of the stethoscope may be placed, day by day, with the same effect. In subclavian murmur, the bruit may change in character several times during one examination; being at one time coarse and loud, at another gentle and musical. The point of maximum intensity may also differ by the variation of an inch laterally on either side.

In aneurism the murmur is permanent. In subclavian murmur the sound comes and goes. It will sometimes stop in an instant, and not reappear for many hours. In one case I knew it to be absent for three weeks, and then suddenly to recur and remain for a period equally long; again to subside, and again to present itself; and so on during many months of observation.

In aneurism, the murmur is not influenced by the movements of respiration. In subclavian murmur, the sound can be intensified by a moderately full inspiration; can often be stopped by a forced inspiration; and, again, can be equally stopped by a prolonged expiration.

In aneurism, the stethoscope produces no change in the murmur, however firmly the pressure may be applied; except in extreme cases, where the tumour

protrudes through the thoracic wall. In subclavian murmur, the firm application of the stethoscope will itself check the exhibition of the phenomenon.

In aneurism, movements of the arm do not influence the sound. In subclavian murmur, the sound can invariably be modified by merely raising the arm, and changing the angle of the limb to the body.

In aneurism, there will commonly be present dulness on percussion over the seat of the pulsation; such dulness being independent of tubercular disease of the lungs. In subclavian murmur, if in the region of the murmur there is any dulness on percussion, the stethoscopic and other physical signs will, in the majority of cases, perhaps in all, show that the deficient resonance is due to tubercular deposition.

In aneurism approaching the surface there may be fremitus; but the fremitus is permanent under all conditions. In subclavian murmur there may be fremitus; but this can be made to disappear by movement of the arm on the side affected, and also by a deep expiration, or a very deep and sustained inspiration.

In aneurism of any of the three great vessels I have named, there will be, as a general rule, symptoms of dyspnœa and respiratory oppression. Subclavian murmur is not, *per se*, accompanied with any such complication.

Finally, as a diagnostic difference between subclavian murmur and subclavian aneurismal murmur, this may be observed; that, if simple subclavian murmur be present on one side, it can usually be detected on the other side; and when the two sounds

are traced downwards, towards the sternum, they are found to be lost, showing that they have no common point of origin, but an origin purely local in each artery. Now, whilst double subclavian aneurism is not an impossible, it is an unrecorded phenomenon, and in common experience is so rare, that the fact of double murmur may be considered as affording direct evidence against aneurism as the cause of the abnormal sounds.

The diagnosis as between subclavian murmur and *cardiac murmur* is comparatively easy. By carrying the stethoscope from the subclavian region to the cardiac region, it will be detected in pure cases of subclavian murmur, that the heart-sounds are both distinct, and that the bruit in the upper part of the chest is entirely unheard at the heart. However loud it may be in its own region, subclavian murmur never intrudes itself on the cardiac music. This line of diagnosis is therefore direct. But there are cases, and I have seen two such, where, together with subclavian murmur, there was systolic cardiac murmur from mitral disease. But here again I had no difficulty in distinguishing the two morbid sounds; for the mitral murmur was permanent, while the subclavian murmur was intermittent; the mitral murmur was prolonged, while the subclavian was short and sharp; and lastly, on carrying the stethoscope from the subclavian space downwards, towards the heart, an intermediate point was found, where the subclavian murmur was lost and the cardiac murmur began to be

heard; and on the contrary, on carrying the stethoscope upwards, from the heart towards the subclavian artery, an intermediate point was found, where the cardiac murmur was lost and the subclavian murmur began to be heard. There were thus distinctive evidences of two seats for the two murmurs; and the same evidences would, I presume, be present in all similar complicated cases. I had, again, another example, where subclavian murmur existed with loud aortic murmur, and this latter murmur was in truth reflected nearly into the subclavian region. But several differences were detected on careful inquiry, by which the diagnosis was determined. For example, the two murmurs had each a different sound; the subclavian murmur could be stopped by inspiration, by the movement of the arm, and by pressure of the stethoscope; while the aortic murmur was persistent in all these conditions; and a last but excellent point was, that the radial pulsation, which was *synchronous* with the subclavian murmur, *followed* the aortic murmur. I do not think there could be two better marked examples of subclavian murmur, complicated with aortic and mitral murmurs, than those above given. Without further comment, they supply in themselves all the differential diagnostic points to which it is necessary to call the attention of the reader.

Pleural friction-sound may be mistaken for subclavian murmur, or the murmur may be mistaken for friction-sound, but not if care is taken. One of my

pupils once showed me a patient who had, he believed, the subclavian murmur; but, on re-examination, the sound proved to be frictional. The character of the sound was certainly closely analogous to a sharp subclavian whiz, and the position was right for such an occurrence. But the diagnosis was clearly in favour of pleuritic friction: for (*a*) the sound heard was only present during inspiration, and was but one sound in the full period of inspiration; the arterial sound would have been repeated as frequently as the pulse. (*b*) The sound heard was not modified by the position of the arm. (*c*) The sound was still heard when the stethoscope was applied with sufficient firmness to stop the arterial pulsation altogether, and to check the radial pulse.

These three distinctions were in themselves sufficient, as physical signs, to indicate the nature of the case. To them, nevertheless, were added other general symptoms; viz., pleuritic pain increased by a deep breath, a small irritable pulse, and feverish exacerbations. Such symptoms, truly, were compatible with subclavian murmur and tubercle; but, coupled with those which are already named, they completed the evidence, and rendered an almost perfect diagnosis quite perfect. In any other analogous case, similar diagnostic rules would be afforded, in part at least, if not in the whole.

I have met with one case in which, together with subclavian murmur, there was, with chronic bronchial inspiration in the right subclavian region, a sharp

bronchial coo immediately behind the artery. The patient was young, twenty-three years old, had suffered long from bronchial cough, and at the time of my first seeing him had been losing flesh and spitting blood. I was undecided as to the concurrent existence of tubercle; for a sharp bronchial coo at each inspiration covered the physical indications, even if present, of this deposit. At the point under the clavicle where the bronchial sound was heard, there could also be brought out during a deep inspiration a sharp subclavian murmur. But the distinction between the murmur and the bronchial whistle was clearly to be distinguished. In tone and timbre they were nearly the same, but in time they were different; for while the bronchial sound was as one to an inspiration, the subclavian ran on to three, both kinds of sound being heard as distinctly as two parts in a musical scale; added to this, there was the influence of differing positions of the arm, which modified the subclavian murmur, and did not modify the bronchial coo: while, again, the effects of firm stethoscopic pressure stopped the arterial, but in no way affected the bronchial sound.

This case clears the way for diagnosis between the arterial and the bronchial cooing murmurs.

Absolute Diagnosis. The absolute diagnosis of subclavian murmur is as follows.

It is a murmur beneath one or both of the clavicles, confined to the subclavian regions, and synchronous with the pulse.

The murmur is coarse and loud—a rasp; or sharp and musical—a whistle; or soft and musical—a coo; or shot-like, coming down on the ear bluntly and dead.

The murmur is always to be arrested by pressure, sufficient to check the pulse at the wrist, made by the stethoscope on the subclavian artery.

It is susceptible of modification by the movements of respiration. A moderately full inspiration may develope it; a very deep inspiration may either intensify it or stop it. A prolonged expiration will often remove it.

Movements of the arm modify the sound. The position of the arm when it is nearly down by the side of the patient gives, mostly, the minimum of intensity. The position of the arm slightly raised above the right angle to the body is that in which the maximum intensity is usually gained; while movements between these extremes produce varying gradations of both the quality and the frequency of the murmur.

In extreme cases there is to be felt, on application of the finger over the artery at the point where the murmur is heard, a marked fremitus, of which the patient may be conscious as well as the operator.

RELATIONSHIP OF SUBCLAVIAN TO COMMON ANÆMIC MURMUR.

It will be seen, by reference to the table on page 8, that of the 2000 cases from which that record was compiled, there were 10 instances where the disease is classified as anæmia. In these cases there

were bloodlessness and debility, but no physical indications of disease or consolidation of the lung; while, in the cases of murmur in the phthisical and bronchial diseases, the murmur was intensified where anæmic concomitant symptoms were most obvious. I have explained the possible cause of this in a previous page. I am anxious now to state that this fact does not of necessity connect pure subclavian murmur with those examples of anæmic murmur in the neck and heart, which we so often find in children, and in young women suffering from extreme anæmia or chlorosis. I have, indeed, met with no case of pure subclavian murmur in a child, and with but three instances in the female subject. Moreover, in all cases of pure anæmic murmur, whether cervical or cardiac, I have for the last two years sought for the subclavian murmur, but have never met with it as peculiar to such cases. The examples of anæmia from which this inference is drawn amount to somewhat above two hundred. The subclavian murmur, therefore, may be considered as independent of the common anæmic murmur. The two may coexist; and I have shown that the subclavian murmur is intensified by an anæmic condition of blood; but the connection does not extend further.

It is also to be observed that common subclavian murmur exists, as a general rule, without analogous murmur in any other part of the arterial system. But I have met with an exception to this rule. In one case of intense hypertrophy of the heart with mitral regurgitation, there was loud arterial murmur

in every spot where the course of a large artery could be superficially traced. In this instance there was murmur in the axillary artery, in the brachial, in the femoral in Scarpa's space, and in the popliteal. At all these points there was likewise fremitus, from vibration of the vessel, and rapid irregular pulsation, of which the patient was so conscious, that, without producing absolute pain, the vibration was to him a perpetual annoyance, and added greatly to his anxieties and sufferings.

BEARINGS OF SUBCLAVIAN MURMUR ON PRACTICE.

The existence of pure subclavian murmur supplies no special knowledge in regard to treatment. But, in diagnosis, it has much worth when carefully and judiciously studied. Unless caused by mechanical occupations, it indicates the existence of some pressure exerted on the artery by a structure posterior to the vessel—in most instances, we must infer, by the lung. Thus it affords one diagnostic indication both of bronchial disease and of tubercular deposit. The evidences of such amount of bronchial disease as is sufficient to produce the murmur, are sufficient also of themselves to express the extant mischief, without the superaddition of the murmur as a sign. But in phthisis the murmur may prove one of the earliest indications of tubercle. In two cases, where there were general symptoms of phthisis and a phthisical history, but where the ordinary physical signs of the disease were utterly wanting, I was supported strongly in the suspicion of actual tubercular

deposition by the prominence of a subclavian bruit. In both these cases the suspicion was confirmed by after events: for one ended rapidly fatal from tubercle, commencing in the position which the murmur indirectly pointed out; and the other, although the patient still lives, passed on to true tubercular disease of the apex of the lung behind the murmuring artery. The arterial murmur may thus be important, both in regard to diagnosis and prognosis: but it must be taken for no more than it is worth, since it may occur, as we have seen, dependently on various other external producing causes, and may possibly in some examples be produced by a light stethoscopic pressure on an artery thrown slightly towards the surface. With all these objections to it, its presenee ought, notwithstanding, to have its diagnostic value in doubtful cases of tubercle in the apex of the lung.

But the point of most importance in regard to the murmur, is to know it in such perfect manner as not to mistake it for aneurismal murmur. I have shewn the fact of such error, and its consequences; and if this history shall prevent the recurrence of the like mistake, one at least of the main objects with which the essay has been written will be attained.

ESSAY II.

ON A DISEASED CONDITION OF THE NAILS.

THERE is sometimes seen in practice a peculiar disease of the nails of the fingers, or of the fingers and toes, which gives to the patient great annoyance, if not suffering. The disease is recognised in France, I believe, under the title "Psoriasis of the Nails"; but in this country it has no designation, in so far as I can learn: it is known, however, to many practitioners. When, in May last, I brought an illustration of this disease before the Medical Society of London, I found that various of the Fellows at once distinguished it, and knew well the difficulties of treating it. Indeed, the disease, when once seen, is so striking, that it is impossible to forget it again.

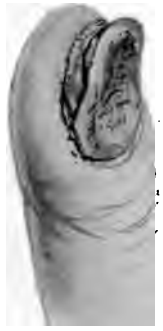
OBSERVED FACTS AND ANALYSES.

In 1856, Mr. C. Housley, now of Port Elizabeth, Cape of Good Hope, drew my attention to the first case I had seen of disease of the nails. He brought to me a patient, who presented the disorder in an intense form. The first indications of the affection in this gentleman had commenced two years before I saw him. His nails became very brittle, and chipped.

After a time, they suddenly became dark on the upper surface, and shining, appearing as though they had been coated with varnish. These changes were unattended by pain. The next indication was, that the surface of the nails began to be pitted—covered, that is, with fine indentations; then the nails were lifted from the fingers by a hard deposit; finally, the raised nails gave way in an irregular manner at the free margins, as though they had been bitten with small teeth.

When this gentleman came before me, every nail in both hands was affected. Some nails were destroyed nearly to the angular junction with the skin, a large and ugly gap being left in place of the nail; other nails were half destroyed, the horny structure being raised a full eighth of an inch from the matrix, and being jagged at the free extremity like the occipital suture. The hands were unpresentable altogether. With all this, the health of the patient was good. There was no eruption of the skin, nor any sign of syphilis, albeit there was an admission of a syphilitic attack some twenty years ago. The hair was firm on the head, but inclined to grey: the age, about forty-two years. The patient himself was engaged as a master in the painting and decorating business. He had sometimes painted with his own hands, but not much. He was a full made, fresh looking man; irritable, I should say, but temperate in living; taking a good animal diet, and a little ale, but not indulging in alcoholics to absolute intemperance. He was dyspeptic, and given to hypochondriasis.

A second case was as follows. A woman, 47 years of age, came before me with chronic bronchial catarrh. In the course of examination, she removed her gloves and shewed me her nails. They were diseased in the same way as in the case immediately preceding. Her history was clearly given. She had been the mother of six living children, and had suffered from one miscarriage. She was still menstruating regularly. Three years previously to the date on which she came to me (April 6th, 1860), she became affected with a scaly eruption, lepra vulgaris, which commenced about the head: there was also some falling off of the hair. I made the most rigid inquiries as to the nature of this eruption and its cause, and no information was withheld me, as I believe; but I could not see reason even for a suspicion of pre-existing syphilitic malady. The lepra continued alone until August 1859, when she first perceived a peculiar sensation beneath the nails. The surface beneath the nails felt benumbed; and at times, especially in the evening, it was the seat of a tingling sensation, described in common *parlance* as pins and needles. The immersion of the hands in water moderately warm would bring out this sensation at any hour of the day; but it always occurred, spontaneously, about bed time. After a few weeks, the nails commenced to look glazed. "They seemed," said the patient, "as if they had been smeared over with varnish of a yellow tinge." They then began to show little indentations all over the surface, as if they had been pricked with the point of a pin, and



had afterwards been rubbed with a black substance. Beneath the nails thickening now began, so that some were raised an eighth of an inch. The edges also crumbled, but not with the same degree of irregularity as was noticed in the first named case.

At the time when the patient came under my observation, every nail on the hands was affected, except that of the left forefinger; the nails of the great toes were also diseased in a similar way. The nails were firmly fixed, to my sensation, when trying to move them; but the patient said that they felt loose to her sensation, and that after soaking them in water they really were loose. Some of them were deeply cupped in the centre. I sent this patient to Mr. Orrin Smith for illustration. He has sketched four of the fingers with great care, together with a patch of the scaly eruption from the arm. The different drawings, given in the accompanying plate, show the nail-disease in various phases, and supply a description which could not be fully expressed in words.

I have only to add, that this patient, though a tall, delicate looking woman, had suffered little illness, and throughout life had been comfortably provided for.

A third example of this form of disease calls for the annexed observations. The patient was thirty-eight years old; a man actively engaged in business, and of genial disposition. He had become stout these last years, much too stout for his time of life, and very dyspeptic. His hair was grey, but firm. He had lived well at all times; and, without being intemperate, had habitually taken *ante horam somni*

a "nightcap," in the form of one tumbler of whiskey toddy. Twenty years ago he had a slight attack of illness, which taught him that Venus not unfrequently transfers her devotees from the sphere of her own charms to the gentle guardianship of Mercury. There was, however, nothing very severe in this attack, and recovery was believed to be complete. In the latter end of 1859, the forefinger of the left hand gave to the patient a disagreeable sensation; the sensation was that of numbness and tingling. The skin then, for about an inch from the tip of the finger, assumed a dry, scaly appearance, with what seemed also to be absolute shrinking of the extremity. The nail became of dirty brown, and slightly pitted; it also wore away at the edges, but not irregularly. The finger was unpresentable, and gave its owner so much annoyance, that he assured me he would rather have it removed by amputation than that it should remain in its existing condition.

The analysis of the cases above cited leave the nature of the disease in obscurity. In the first and last cases, there was a pre-existent syphilitic taint; in the second case, while there were symptoms which are compatible with syphilis, such as lepra and falling off of the hair, all other evidence was against the pre-existence of that disorder. In the first case there was no eruption of the skin; in the last two the skin was affected more or less with scaly disease. The patients were all about the middle age; but in constitutional character the first and last were strongly

opposed to the second. One patient had worked in lead as a painter; the woman had all her life been at domestic duties; and the third was engaged in a mercantile pursuit, in which the pen was more employed than aught else manually.*

CAUSE AND CAUSATION OF THE DISEASE.

The disease allies itself to squamous disorders of the skin, but whether it is identical either with lepra or psoriasis I am not prepared to say; for, while it may exist with these disorders, it may also, as we have seen, exist independently. I think there is no doubt that it commences in the matrix, and that all the conditions of nail are due to a partial or complete death of the structures attacked; in other words, there are interposed between the secreting membrane and the under surface of the nail, epithelial layers, which separate the two structures, and prevent that osmotic change by which the nail is repaired in proportion to its mechanical or natural destruction.

Is the disease purely of syphilitic or of mercurial origin? An affirmative answer has been assumed to this question. Mr. Gay, who spoke at the meeting of the Medical Society, at which some of the cases narrated here were discussed, assumed this affirmative in its entirety, and brought forward, it must be

* In addition to these, I once incidentally saw another case at that period of the disease when pitting with dirty glazed surface are the external symptoms. The history of this case is not known to me accurately; but I may say that the patient was engaged in a profession in which manual labour is excluded.

admitted, considerable evidence in favour of such affirmative. However, I am not prepared to admit the point as proven. I shall show, indeed, evidence further on, which will prove that in some cases treatment negatives the argument of a syphilitic origin. Indeed, in the two examples which I have given, where there had really been an attack of syphilis, I doubt whether that disease was still at work. It seemed to me in both these cases that a free mode of living, and dyspepsia, had more to do with the affection than the specific and long removed enemy.

There is clearly nothing in occupation which leads to the disorder; the first named patient had worked in lead; but I have made inquiries amongst lead-painters, and find that no special affection of this kind is known amongst them; further, we had before us other occupations, all different, and one not manual, so that the evidence points to a constitutional, as opposed to a local, origin for the disease.

DIAGNOSIS.

The disease of the nails under review has three stages. The first stage is marked by a sensation of numbness and tingling beneath the surface of the nail. In the second stage there is an appearance of dirty glazed discoloration of the nail, with pitting of the surface. In the third stage there is a crumbling down of the nail-structure from the edges and the free margin, with more or less of irregularity, thickening from the under part, cupped surface depression, and looseness.

TREATMENT.

In the first case to which my attention was drawn, various forms of treatment had been already pursued. Local treatment had been tried; the nails had been washed with lotions of zinc and lead; they had been encased from the air; and they had been painted with iodine. Internally, mercury had been administered; after that quinine; and again mercury. Believing at first that the disease was of syphilitic character, I suggested iodide of potassium internally in five grain doses three times a day; and my friend who brought the patient to me carried out this treatment fairly for many weeks. It had no effect for good. The patient got tired, and consulted a new practitioner altogether: I have been unable to learn with what effect.

In the case of the woman, the treatment adopted previously to my attendance had again been both local and general. Locally, zinc lotions, zinc ointment, glycerine, and iodine had been applied. Internally, mercury had been administered, and iodide of potassium, but with no avail. The presence of the skin-eruption turned me to another indication. Instead of pursuing local treatment, I commenced with Fowler's arsenical solution as a general medicine, in doses, first of three minims, and afterwards of four minims, three times daily. The good effect of this treatment was quickly shown. The first dose of medicine was taken on the 6th of April. On the 26th of May the eruption of the skin was greatly

better, and the disease in the nails was entirely arrested. On the 26th of July all the nails had nearly regained their normal character, except the nail of the right forefinger. At this visit the patient complained of catarrh; the arsenical mixture was therefore replaced by a saline, which she continued to take until August the 9th without consulting me. At her return on that day, I found the squamous symptoms the same. I once more ordered the arsenical solution; and, as she bore it without any unpleasant sign, it was continued steadily until August 23rd. The nails might now be considered as well. All were restored to perfect smoothness, except the first and second of the left hand. These retained the faintest signs of indentation, and the first one was rather thicker than the others; but an observer, who saw them without previous knowledge, would certainly not conceive that they had been extensively disorganized. The eruption on the skin was yet not altogether removed. There was an irritable patch around the nipple of the right mamma, and a second one in the groin. The medicine was continued until the 1st of September, when the skin eruption had also disappeared. After ceasing the arsenic for a month, there was a reappearance of cutaneous scales about the nipple; but the nails continued unaffected. The arsenical solution was reordered, again with removal of the squamous patch. This patient still remains under treatment.

The third case had been treated merely locally; iodine had been particularly tried, but without any

good result. When the case was in my hands, the first thing I did was to prohibit the "nightcap," and otherwise to reduce the hydrocarbon supplies. Medicinally, Fowler's solution was prescribed, in three minim doses three times a day. After one month, during which the medicine was well tolerated, the nail underwent rapid progress towards renovation; and three weeks later it, together with the finger, had recovered the natural appearances.

The evidence in respect to the treatment of this disease of the nails is thus strongly in favour of arsenic as the remedy. This view in some measure removes the hypothesis of the invariably syphilitic origin of the disorder, and shows, at all events, that there may, as in lepra, be two sources, one of which is not directly of venereal growth.

In any doubtful case, where arsenic alone did not cure, it would be good practice to move from Fowler's to Donovan's solution (the compound of hydriodate of arsenic and mercury), a solution which, notwithstanding Hebra's opinion upon it, is admirable in obstinate cases of mixed syphilitic and squamous disease.

ESSAY III.

ON REDUPLICATION OF THE SECOND SOUND OF THE HEART.

THERE is perhaps no phenomenon more curious to the practising physician, when he first meets it, than that known as reduplication of the sounds of the heart. It is so rare that its novelty strikes forcibly; it is so well marked as a peculiarity when it does occur, that its existence can admit of no dispute.

Those who have not heard what is called reduplication, may learn it by hearing what it is. In lieu of two heart-sounds to each action, there are three; in lieu of the familiar "lūb-dŭp" and pause, there is distinctly caught "lūb-dŭp-dŭp"; that is to say, a third and unexpected short sound takes in great part or altogether the place of the pause. Therefore, in strict definition, reduplication applies to but one of the sounds of the heart.

OBSERVED FACTS AND ANALYSES.

In the beginning of this year (1860), a young man, 23 years of age, came before me at the Royal Infirmary for Diseases of the Chest, with the following complainings. He was subject, and had been subject for

three weeks past, to almost constant palpitation of the heart. He believed that his first symptoms were due to a "cold on his lungs". The palpitation kept him sleepless, and in subdued suffering; he could feel a tilt of his heart against the chest-wall, not in the anterior point, where the beat is ordinarily felt, but laterally—towards the left side. At times this palpitation became unusually violent; there was a sensation of choking in the throat, a struggle for breath, and afterwards great exhaustion. In addition to these signs, there were flatulency and anæmia: but the tongue was clean, the bowels acted daily, and the skin was cool. There was desire for food, but a dread of taking it, inasmuch as anything like a meal oppressed the chest; but the urgent symptoms of which the man complained, and of which he came to be relieved, were the palpitation and oppression. "If I could lie down and sleep", he said, "I should be well; but, as I try to sleep, this terrible weight and palpitation come on increasingly, and I start up at last as if I were being strangled, or crushed between two heavy doors."

The symptoms described by the man might have passed current in the run of practice for those of hysteria and anæmia. Fortunately—if that is fortunate which becomes a habit—I examined the chest of this man. I could find in the lungs no trace of tubercle, bronchial dilatation, nor other organic lesion: yet this was peculiar, and I pointed it out at the moment to Mr. Louis Parnell, who was attending my clinique, that there was everywhere a deficient

respiratory murmur. Turning to the heart, I found it unusually active, and giving a sharp quick thoracic impulse against the chest-wall on the left side, between the fifth and sixth ribs, and rather bearing upon the sixth rib itself. This impulse could be seen as well as the normal anterior impulse, but much more evidently. Each impulse was marked at least eighty times per minute, and without apparent intermittence: yet the action was irregular; for, if the number of beats were counted one minute, and then again for a second minute, there would be eighty in the first minute, and eighty-five in the second; but if each beat were taken singly, there was no apparent irregularity between them, the disparity in the whole number of beats per minute being distributed with an equal gradation, extending over some measurable period of time, whether towards increase or diminution.

On taking the sounds with the stethoscope, there was to be heard, with unceasing regularity and clearness, the reduplication which I have already briefly noticed—lūb-dŭp-dŭp, lūb-dŭp-dŭp.

I ordered for this man tincture of the sesquichloride of iron in ten minim doses, in bitter infusion, twice a day. The symptoms, it will be seen from this treatment, were supposed to be mainly dependent on anæmia and dyspepsia: we shall find in the end how far that suggestion was right.

Three days after this treatment had been carried on, the man came to me again. He was irritably nervous; and, as I thought, gave to every symptom

an exaggerated colouring of the sombre tint. The medicine did him harm, was his argument; it did not confine his bowels, but it made him worse every dose—of that he was sure. Not believing much of this report, I nevertheless changed the individual remedy, but not the direction of the treatment. I ordered him a combination, which I have found most efficient in many cases of dyspeptic anæmia; namely, trisnitrate of bismuth in ten grain doses, with five grains of the ammonio-citrate of iron and an ounce of water. This medicine was persisted in twice daily for a week without change. The oppression was complained of as before; and the reduplication was as clear as ever. I now added to the mixture a little sulphuric ether, in fifteen minim doses, and ordered a five grain galbanum pill at bedtime. The next week my man did not come; nor the next. I feared he was lost as a history, and regretted it much. But, five weeks after he had left me, he reappeared, very comfortable in health, rather triumphant in manner, and entirely relieved of reduplication and palpitation. His relief occurred thus.

On the second night after his last visit to me, he went to bed very oppressed; he was restless all night, often getting up and going to the window for air. He lay down more composedly towards the morning, and, worn out, fell asleep. Soon he awoke; and his wife and friends were truly and naturally alarmed at his appearance. The oppression at his chest prevented respiration; his lips were blue; his limbs convulsed. He was with difficulty half-dressed, put

into a cab, and conveyed to one of our hospitals. Having arrived there, he was, either by a rule of the institution, or at the instance of an attendant, put into a hot bath. As his body became warm the oppression ceased; or, to use the patient's own words, "as my skin got hot and red, the distress rose, as it were, from me altogether." He stepped out of the bath into a warm blanket, went to bed, perspired freely, and rose next day as well as he ever was in his life. He was kept in the hospital a few days, and took a dose of castor oil, but nothing more. He told his physician that he had been under previous treatment, and that he believed his heart was affected. The physician listened to his heart, said there was nothing there wrong, and dismissed him cured.

I have met with two other cases besides this of reduplication; but in neither was the phenomenon clearly developed: it was fleeting and faint. In one of these cases, the patient was a man, who was phthisical; in the other, a woman, also phthisical. I lost sight, unfortunately, of both cases after they had been once or twice before me.

CAUSE AND CAUSATION OF REDUPLICATION.

The cause of reduplication of the heart-sounds has been most carefully studied by my friend and colleague Dr. Cockle, who has met with many cases; and whose explanation of the phenomenon varies but little from my own. The idea of cause can only be arrived at by considering each position of the question separately.

The first point to be settled is, whether the reduplication, as it occurs, is a reduplication of the first or of the second normal sound. I think no one who is accustomed to physical diagnosis could have doubted that, in the case I have given at length, it was the second sound that was repeated. The added sound was the same as the second in quality of tone —dŭp; while the first normal sound occupied its true position in regard both to time and to quality. The added sound, again, occupied the position of the natural pause, yet not entirely; for the briefest pause could be detected when the action of the heart was quieted by a forced and prolonged expiration. The pause thus discovered was found to occur at an interval between the reduplicate sound and the first sound.

Again, notwithstanding the double sound, the pulse was not intermittent, but was presented, as is natural, upon the systole of the heart coincidently with the systolic sound. This showed that the systole was perfected at one stroke on the arterial side, and that the aortic valves came down after the systole in correct time, and without suffering regurgitation of blood through them.

Lastly, the double sound was most prominent over the aortic and pulmonary orifices: lower, towards the apex, the first or systolic sound was, as is usual, most distinct.

In the other two cases to which reference has been made, the same proofs existed to show that the fact of reduplication was with the second or diastolic

sound. If, then, this point be proved, we must consider how the second sound admits of the modification which we have seen.

That the second sound of the heart is produced by the tension of the semilunar valves, is a fact which no one who has studied the subject physiologically can logically dispute. As little ought any one to dispute that the first sound is the product of the tension of the tricuspid and mitral valves.* But, this latter point set aside, it is sufficient here to urge that the second sound is the result of the common tension of the aortic and pulmonary semilunar valves. When, therefore, this sound is doubled, as in the case above stated, the added sound must be due to one of three sources.

It must be a reverberation of the second sound:

It must be a repeated second or diastolic sound *in full:*

Or it must be produced by a want of simultaneous action in the aortic and pulmonary semilunar valves.

The first of these suggestions is untenable, because there is no known acoustic law by which, in the closed circuit of the blood, a reverberation could be explained.

The second hypothesis is untenable, because the reduplication is confined to one sound; for, to produce a double simultaneous tension of the semilunar valves, there must be double ventricular action, and

* See, who will on this question, the overwhelming arguments of Dr. Halford, in his Physiological Essay on the Heart-Sounds—an essay which reduces the action and sounds of the heart to mathematical demonstration.

therefore a double systolic as well as diastolic murmur, in the period of one action of the heart; which is impossible.

We are driven thus to the third explanation, which seems, indeed, to answer every purpose. Thus, if in any given case there is set up an obstruction to the current of blood from the right auricle to the lungs, the current through the aorta from the left ventricle being free, the left ventricular systole will be made before the right ventricular systole; and the obstruction in the pulmonary circuit will prevent, for a period abnormally long, the return contraction of the pulmonary artery, and the closure with tension of the right semilunar valves. The two sets of semilunar valves will in this way be prevented from acting simultaneously, and the common sound produced by their tension will not be struck in the same period. The aortic valves coming down first, under these circumstances, will strike one diastolic sound, in the normal period of the diastolic sound; the pulmonary valves, lagging behind, will come down later, and strike a second sound in the period of the pause; and then the systolic sound will follow in its natural place, and in its natural intensity. This was, I believe, the order of events in the case which forms the text of this essay; and the same are the events, I presume, in all cases of reduplication.

The same view that accounts for the physical cause of reduplication, accounts also for the general symptoms which accompany the physical phenomenon. The balance of the circulation is disturbed in

these examples, and the results are obvious. The oppression of respiration, amounting, as it did in my cases, to partial asphyxia, indicated with sufficient clearness the embarrassment of the pulmonary circulation; the deficient respiratory murmur defined the same fact: the palpitating heart indicated the labour demanded to support two cardiac currents unfairly balanced; and the inability to sleep free of disturbance showed the perpetual necessity for constant and undiminished action of the forces of respiration and circulation to meet the difficulties in their way.

But that which most of all indicated the nature of the reduplicative phenomenon in the text case, was the result of the treatment. Let us mark the fact specially. As soon as a powerful derivation was set up by the hot bath; as soon as the vessels of the skin, expanding under the heat, claimed more blood, and so relieved the lungs; so soon was the pulmonary and systemic balance restored, and the patient cured. He felt, indeed, as he expressed, that the oppression which had wearied him for weeks passed from him altogether.

DIAGNOSIS OF REDUPLICATION.

I can recall no other abnormal sound, which could be confounded with that of reduplication. The absolute diagnosis is as follows: a physical condition of the heart during which, on listening to the cardiac sounds, three sounds take the place of two, the last of the three occupying, or nearly so, the period of pause.

The added sound, during ordinary respiration, fills the place of the pause partly or altogether. The sound is that of the diastole or second sound; short, and with an accent, dŭp. It is heard most distinctly over the position of the pulmonary and aortic orifices. It is attended with symptoms of increased and irregular action of the heart, oppression of the breathing, and feeble respiratory murmur.

I have, so far, treated the subject of reduplication of the sounds of the heart with special reference to reduplication of the second sound; and this for the reason that as yet I have never met in my practice with any case in which the added sound could be considered as connected with the true systolic sound. It is but fair to say, however, that most competent observers, amongst them Dr. Cockle, Dr. F. C. Webb, and Dr. Walshe, have met, as they believed, with illustrations of systolic reduplication.

In these examples, the character of the abnormal sound must stand as lūb-lūb-dŭp; and the cause of the additional sound must be due to the circumstance that the ventricular action of the two ventricles is not synchronous, and that, as a result, the tension of the tricuspid and mitral valves is not produced at the same instant. Now, while this event of irregularity of tension between the tricuspid and mitral valves is possible, and is, I believe, occasionally the fact, it is difficult to explain how by such occurrence a double first sound could be heard *with a single second sound*, seeing that the second sound itself is not a mere follower of the first, but is dependent for its

production primarily on the systole. For unless either ventricle contract with sufficient force to distend the artery emerging from it, aortic or pulmonary, there can be no resistance, and no back stroke to close the semilunar valves and produce a second sound; and, as it is impossible that ventricular contraction can take place without tension of the auriculo-ventricular valves, and, therefore, without a systolic sound, so it would seem equally impossible that a first sound should occur twice without a second sound following immediately upon it. A condition such as is described would then give a double first or second sound in the period of one arterial pulsation—a phenomenon which, I believe, has never been met with.

At the same time, there are conditions, as I have before said, when the ventricular walls being greatly weakened and irregularly weakened, there is an irregularity of contraction. Here, however, the auscultatory phenomena are of two kinds, according to the degree of feebleness of the two contracting ventricles; in one of these cases one ventricle contracts with sufficient force to produce valvular tension, while the other produces but feeble tension, and the effect is a weakened systolic sound; in another set of cases both ventricles contract so feebly as to produce imperfect valvular tension on both sides, the effect being to produce a confused rustle, in which no systolic sound can be detected, and often but a feeble second sound. We meet with this frequently in cases of large disorganized heart, when the mus-

cular structure has undergone extensive degeneration, with thinning of the parietes.

From these observations, I should infer that what is considered a repeated first sound is, in fact, the first intensified stroke of a double second sound; but I would not venture to offer these remarks dogmatically, and as necessarily true, although the explanation carries to my own mind the conviction of a truthful rendering of a difficult problem.

BEARINGS ON PRACTICE AND TREATMENT.

The study of those cases in which reduplication of one of the heart's sounds is detected, opens to us various considerations having relationship to other affections marked by analogous general symptoms.

I believe that in many of these cases, where there are symptoms of palpitation, oppression of breathing, especially during sleep (nightmare), and irregular pulse, the symptoms, usually attributed to indigestion, are due to a disturbed balance between the pulmonary and the systemic circulations. This want of balance may arise from different causes: from temporary congestion of lung after an undue supply of food to the system; from irregular contraction of the heart under the influence of disease affecting one or other ventricular wall; from pressure on the diaphragm by collection of flatus in the stomach and intestines.

Treatment. There is nothing more obvious than the fact that, in the treatment I pursued in the case which affords the subject of the present commentary,

I was entirely on a wrong track. The man was correct when he said that he was made worse by the medicines (tonics) which were prescribed for him. They suppressed secretion, which was the physiological act requiring more excitation; they confined the current of the blood, and, in proportion as they did so, they supported the disturbance in the circulation.

It is equally obvious that the treatment by the hot bath, given though it might have been almost by accident, and certainly without any idea of relieving an embarrassed right ventricle, was the rational, as it was therefore by necessity the successful treatment. Any other form of derivation would have given relief—the abstraction of a little blood, a sharp purge, or diaphoretic; but the warm bath was and is the simplest remedy.

I have learned much from this case of reduplication; the essence of all which is to the effect that whenever, without evident sign of disease of the lungs or of organic changes in the heart or nervous centres, there are manifested indications of vascular irregularity commencing at the heart itself; then, however clear may be the signs of anæmia and debility, no good will come from any treatment in the absence of some system of derivation; that tonics, administered alone, invoke more mischief; and that coincident debility disappears without them, when the troubled heart is set at liberty.

ESSAY IV.

CONTRIBUTIONS TOWARDS A MORE PERFECT CLINICAL HISTORY OF SCARLET FEVER.

For some years past, I have been directing particular attention to Scarlet Fever, with a view to bring forward certain facts tending to supply a more correct clinical history of this, as yet, formidable disease. Some of my labours in this direction have already been laid before the members of the Epidemiological Society, and have appeared in abstract in the periodical literature of the day. I propose, on the present occasion, to collate these scattered materials, and, with certain new observations, to condense into one clinical essay the result of all that I have as yet accomplished. It is little enough, I am well aware; but such as it is, it is herewith offered.

POINTS RELATING TO THE NATURAL HISTORY OF SCARLET FEVER.

The question of the *occurrence of scarlet fever at different periods of life* is one of much interest, and has occupied the attention of various observers. Regarding this, an opinion has been promulgated that the disease is rare, if not absent, in babes at the breast and

in children under two years of age. This statement was, I believe, first made by the distinguished Withering; but Dr. Nieuwenhuys, of Amsterdam, in an admirable paper on small-pox and scarlet fever, has repeated, within these late years, an almost similar opinion. He seems to have been led to this view by seeing cases in which infants had escaped the disease whose mothers were suffering from it, and were occasionally suckling these same children during the period of the disease. If this statement were true, it would convey one of the most curious facts in medicine. It is, however, one of those errors which occasionally will creep into the works of the ablest writers, if they give the history of their individual knowledge alone.

It happened to me that, in the first case of scarlet fever I ever saw in a puerperal woman, the infant at her breast died of the disease, and that another child of hers, under two years, took the malady and suffered from it in the malignant form. Here, then, were facts at once offered which showed that no rule such as that which has been suggested really obtains, or at least that such rule had its exceptions. I thought, therefore, that it would be interesting to follow out the subject, and, from a rigid analysis, to learn the fatality of scarlet fever in the early terms of existence.

In this direction I took, in the first place, my own reports of cases of the disease, and collected from the printed return-books of three friends who held parochial appointments (viz., the late Mr. Brown, of Saf-

fron Walden, in Essex; Mr. Palmer, of Sheen; and Mr. Beresford, of Narborough, Leicestershire), the cases there reported from the year 1849 to 1853. To these I added statistics from the accounts of Messrs. Ryland and Parsons, who observed and noted the disease in Birmingham from the year 1832 to 1835. The result of this work gave me 431 cases of the disorder. Of these cases, 329 were in children under ten. In 242 cases under five, there were no less than fifteen in infants under two years of age.

To pursue the question further, I next turned to the mortality tables of the Registrar-General. The following is an epitome of this research:—

"Out of a total of 12,962 deaths from scarlet fever in children under five years of age, occurring in the county of Kent in 1843, in London in 1845, 1846, and 1848, and in England in 1847, there were—

Age.	No.	Age.	No.
Under 1 year . . .	1,289	Over 2 and under 3 . .	3,338
Over 1 and under 2 . .	2,874	Over 3 and under 4 . .	3,063
		Over 4 and under 5 . .	2,398
Total under 2 years .	4,163	Total from 3rd to 5th yrs.	8,799

Total from 1 to 5 years, 12,962.

Again, "in a total of 3,795 deaths in patients under five years, occurring in the London hospitals, in Manchester, and in Birmingham, in the year 1839, in twenty-four towns in 1840, and in London in 1842, there were—

Age.	No.	Total.
Under the second year	410	3795
Over the second, and under the third . .	1797	
Over the third, and under the fifth . .	1588	

While it is thus clear, from indisputable evidence,

that the idea of an immunity from scarlet fever in the earliest period of life is founded in error, it comes out, also, from a more extended inquiry, that the mortality from scarlet fever is actually greatest in the period of infancy, if that be extended from one to five years. Thus, out of 31,744 cases of scarlet fever, occurring in Manchester in the year 1839, in Liverpool in 1839, in Birmingham in 1839, in twenty-four town districts in 1840, in London in 1842, 1843, 1844, 1845, 1846, 1848, and in England in 1847, the ages were—

Total number of deaths.	Under 5.	5 and under 10.	10 and under 20.	20 and under 40.	40 and upwards.
31,744	21,469	7,756	1,755	552	212
Per cent.	67·63	24·43	5·52	1·73	0·66

We gather from these details, as an exposition of our first inquiry, that scarlet fever attacks most frequently in the third and fourth years of life: that it declines rapidly after the fifth year, and is almost lost after the fortieth. At the same time it would be unfair to attribute to age any direct relationship to the cause of the malady: age cannot be looked upon either as predisposing or preventive. It is due to the non-recurrent character of the disease after one attack, and to extreme susceptibility to it of all who have not suffered, that the ratio of cases at different ages is so marked. Children of three or four, who are by that time permitted to run about, and are also susceptible to the disorder, are naturally more subject to exposure than infants on whom the mother's eye is constantly watchful; the former, therefore, suffer most. And for the reasons that the majority of cases take

place early in life, and that the disease is non-recurrent as a general rule, children over ten, and adults, are, to an extent proportionately large to the whole population, protected from the disease.

Leaving the question of age, I find that great differences of opinion have been offered on the point, *whether sex exerts any influence on the occurrence of scarlet fever?* The prevailing idea on this subject has been, and is, to the effect that females suffer most. Dr. Tweedie, whose opinion we all recognise, arguing on his own general experience, and on the results of an imperfect table, says, "females are more subject to the disease than males": with great candour, however, he repeats the opinion of Withering, "that, in children, the numbers of sufferers are equal in both sexes; but, amongst adults, females suffer most." Fothergill, in writing of the malignant attack of scarlet fever which visited London in 1749, says, "a greater number of girls have it than boys, more women than men." Rayer affords similar testimony; but Dr. Binns, in describing an epidemical scarlet fever which occurred in the Ackworth school, tries to show, by reference to a small but, as far as it goes, fair table of cases, that girls are less liable to the disease than boys.

The 431 cases in my own possession, and to which I have before drawn attention, show an equality between the sufferers of different sexes. I could not, however, consider so few cases as 431 worthy of note for any definite conclusion derivable from them.

Once more, therefore, I turned to the Registrar General's reports, and gathered what succeeds.

In 102,382 deaths from scarlet fever, occurring in England in the years 1838, 1839, 1840, 1841, 1842, 1847, and 1848, and in London in 1842, 1843, 1844, 1845, 1846, there were—

Amongst males, 51,660 }
„ females, 50,722 } = 102,382 (938 males in excess.)

This calculation took in sufferers of every age, and left, at first sight, a fair inference on the mind, that males are, on the whole, more subject than females to scarlet fever. To understand the question fairly, however, it was necessary to go further; viz., to analyse a large number of cases, and find at what *ages* the deaths occurred.

34,744 cases of death from the disease, occurring in Manchester, Liverpool, and Birmingham, in the year 1849, in twenty-four towns in 1840, in London in 1842, 1843, 1844, 1845, 1846, and 1848, and in England in 1847, were therefore analysed. The relative number of deaths in both sexes, and in certain specified periods of life, were—

Age.	M.	F.	Total.	Predominance.	
				M.	F.
Under 5 years	11097	10372	21469	725	...
From 5 to 10	3927	3829	7756	98	...
„ 10 to 20	844	911	1755	...	67
„ 20 to 40	261	291	552	...	30
„ 40 and upwards .	97	115	212	...	18
Total	16226	15518	31744		

From this calculation, I derived the general fact that, under the age of ten, more males die from scarlet fever than females; but that, above ten, the contrary obtains.

Having ascertained this preliminary fact, I looked at the question in its bearings on relative mortality in reference to relative population. It was very difficult to find data of any extensive kind to elucidate this relationship; but ultimately I got one district where population and special mortality admitted of being studied together.

In the districts of Kent, in the year 1843, there were—

Deaths from scarlet fever at all ages, 413, of which	205 were males 208 females	giving 3 females in excess.

The population of these districts, as obtained by the census taken rather more than a year before, was—

Total population in 1841, 469,113, of which	232,228 were males 236,885 females	giving 4,657 females in excess.

Now, this last table is, it will be seen, quite contrary to the first two in its results; and separate writers, inclined to controversy, might, by referring to only one of these tables, baffle an adversary considerably. Considered, indeed, in their singleness, the tables might and would be made to convey the most incorrect ideas. But when the three tables given are viewed carefully, and with an unbiassed eye, their very differences are found to be of the highest value, inasmuch as these differences place before the mind the true state of matters. For, *collectively*, the cal-

culations indicate that scarlet fever makes no selection as regards sex, but attacks more males, or more females, *according to the relative number of males or females who are resident in any district where it is epidemical.*

The last table illustrates this fact exceedingly well. There are more deaths from scarlet fever amongst females in a given portion of country than amongst males; and there are in the same portion of country, previous to the searlet fever epidemic, a larger number of females than of males in the population.

Again, the calculation given at page 58, which shows that scarlet fever is most prevalent in the first ten years of life amongst males, and that after ten the preponderance passes over to the female side, illustrates the same law. For, in the child part of our population—using the word population in its general sense—males number most; but in the youth and adult population, there are more females than males, owing to the fact, that youths and men are exposed to more causes of mortality, and are drafted into other countries in larger numbers, than girls and women: so scarlet fever, as the results given above illustrate, follows this law of number; for our tables show, that whilst more males die under ten years from the disease than females, after the age of ten the mortality begins to take the lead on the female side, and to continue steadily in the same course as life advances.

Still more, our calculation from the 102,382 cases

with which we opened this argument, although it gives a slight excess on the male side, viz., 938, supports the same law. For the majority of these cases occurred in patients under ten years; in the period of life, that is to say, when the body is most susceptible of the disease, and when the male population is larger than the female.

I think I have put down sufficient now to show that sex has no influence, predisposing or otherwise, on scarlet fever; but that male and female are alike susceptible if they are alike exposed.

The influence of meteorological conditions in relation to scarlet fever is a next point of peculiar interest. In the years 1852-3, I made extensive inquiries touching this matter, with special reference to barometrical and thermometrical changes, the presence or absence of elasticity, the direction and force of the wind, the humidity of the atmosphere, and the effect of season on the course and prevalence of the disease.

The only reliable fact affirmative in kind, as bearing on the effect of meteorological conditions, was in relation to season only. Here I arrived at valuable information which bore out, in the strictest sense, the opinions of preceding writers. The concurrent testimony, in fact, of almost all writers on the disorder, teaches that it is most common in autumn, next so in the summer, next so in the winter, and least so in the spring. In three epidemics which I witnessed in different parts of this country, the disease always commenced in the summer, became

most violent in the last months of the year, continued into the new year, and died away with the spring. I believe, too, that sporadic cases are most common in autumn, although I have seen such instances in the month of April. Cases, however, occurring in the spring, even though of malignant character, seem to me more likely to recover than at the other seasons.

Sydenham remarks that scarlet fever, though it may occur at other seasons, is most common in the end of summer, when it attacks whole families, and children especially. Fothergill, in describing the malignant form of scarlet fever, observes:—"Although it survives different seasons, and all varieties of weather to which we are exposed, yet it seems to show itself most frequently in the autumn, and in the beginning of the winter; at least I have met with more cases from September to December inclusive, than in all the other months together."

Cullen names the beginning of winter as the time when scarlet fever is most prevalent. Withering speaks of the winter and summer months as favourable seasons, and records the particulars of an epidemic which, commencing in summer, was temporarily checked in October, but recommenced, with extreme virulence, in November. The terrible epidemic of malignant scarlet fever that raged in St. Albans in 1748, is described by Dr. Cotton as occurring in the latter end of the year. The epidemic recorded by Peart in 1802, did not become violent and extensive

until the latter part of August, and in September. Willan remarks that the disease, scarlet fever, is most virulent during October and November. Haygarth entertained a similar opinion. Rayer observes that the disease is most common about the equinoxes. An epidemic of malignant scarlet fever, described by Chomel under the title of gangrenous sore throat, and which raged in Paris a hundred years ago, was most violent in the months of October and November. Dr. Nieuwenhuys, in his paper on "the scarlet fever in Amsterdam in the year 1834," remarks, "the disease first showed itself, in the epidemic form, in the month of June 1834, and its victims were—in June 22, in July 32, in August 50, in September 78, in October 136, in November 106, and in December 61."

Mr. Ryland, in describing the cases of scarlet fever which occurred under his care amongst the out-patients of the Birmingham Infirmary in the year 1835, states that in the first quarter of the year ending March 25th, he had 5 patients, in the second quarter 7, in the third quarter 35, and in the three last months 59, making 106 cases in all.

Finally (many other authors being omitted), Dr. Tweedie, in his essay on scarlet fever, states that the disease is most common in autumn, least so in spring.

Such are some of the conclusions to which general observers have come with reference to the prevalence of scarlet fever at the various seasons of the year; and when these conclusions are tried by statistical facts, their correctness is strikingly established.

In 46,077 deaths from scarlet fever in England in 1841 and 1842, and in London in the years 1838, 1839, 1840, 1843, 1844, 1845, 1846, 1847, and 1848, the proportions of deaths in the seasons of those years run as follows:—

	April, May, June.	July, Aug. Sept.	Oct. Nov. Dec.	Jan. Feb. March.	Total.
No. of deaths...	9,068	11,914	14,630	10,465	46,077
Percentage.....	19·679	25·856	31·751	22·711	

The above table requires no comment. Derived from an immense number of cases, the inference to which it leads, viz., that scarlet fever is most prevalent in this country in the last three months of the year, least so in the months of April, May, and June, is obvious. One word I must add. If the reader could see an analysis of the above tabular statement, he would find that the rate of mortality is not always the same in the special quarters of the years specified. So that a calculation made from one or two, or even three, of those years would be likely to lead into a grievous error. When, however, the trouble is taken to reduce the returns of a great number of years to a single calculation, we approach as near to the truth as absolute knowledge can carry us.

The correctness of these results is, moreover, singularly confirmed by the calculations of Dr. Tripe. In a paper in the *Transactions* of the Epidemiological Society for 1857, Dr. Tripe takes up the statistics of scarlet fever from 1840 to 1856, as they are recorded in the returns of the English Registrar-General. The

summary of Dr. Tripe's labours in this direction was as follows:—

"Of the total mortality in the years 1840-56, viz., 33,451, 6042 deaths happened in the spring, 7910 in the summer, 11,706 in the autumn, and 7793 in the winter quarters; or in the following ratio, 18.0 per cent. in spring, 23.6 per cent. in summer, 35.2 per cent. in autumn, and 23.2 per cent. in winter. This disease is, therefore, by far more fatal in autumn than in any other season, and, as before shown, rages most furiously from the middle or end of September to the middle of November; the largest number of deaths having occurred in October. I put the percentages in a tabular form.

Percentages of Deaths from Scarlet Fever.

Years.	Spring.	Summer.	Autumn.	Winter.
1840-56	18·0	23·6	35·2	23·2
1840-49	17·7	24·8	35·6	21·9
1850-56	18·5	22·2	34·3	25·0

"The variation in rate of death in the quarters of the two periods under examination was much less than in either measles or small-pox. In the spring quarters of 1840-49, the rate was 17·7, and of 1850-56, 18·5 per cent.; in the summers of 1840-49 it was 24·8, and of 1850-56, 22·2 per cent.; in the autumns of 1840-49 it was 35·6, and of 1850-56, 34·3 per cent.; in the winters of 1840-49 it was 21·9 per cent., and of 1850-56, 25.0 per cent. From these data it is evident that the greatest mortality from scarlet fever occurs in autumn, and the smallest in spring. The variation in the rate of mortality was greatest in the winter quarters; for in the winters

of 1840-49, out of each 100 deaths, 21·9 per cent. occurred, whilst in 1850-56 no less than 25·0 per cent. happened.

"The rate of death in the two periods was by no means alike; 1858 deaths having been registered in each year of 1840-49, and 2124 in each of the years 1850-56, being an excess of one-seventh. This has arisen from the disease having assumed an epidemic form at shorter intervals than usual."

Now, in considering season and its influences, what are we to interpret from it? The term is too wide to be in any degree definite as a cause of disease. What is there in season to determine the origin and modify the course of a pestilence such as scarlet fever? I confess I am unable to tell; for, when the broad affirmative fact is established, all in the way of detail is negative.

I did, indeed, once with great labour endeavour to analyse the facts bearing on this question. I sought through the meteorological changes of season for an explanation of the prevalence of this one disease at particular periods. Was there anything in temperature, in barometrical pressure, in rain, in the electrical conditions of the air, and, above all, in the movements of the winds, that could influence the course of the disorder? It was a painful failure, this research; and I will show it in its failure. After various endeavours to arrive at results which should be to my mind satisfactory, I determined at last to find certain periods when the disease in one given city, say London, should present in a given time, say a

week, the same rate of mortality. This, as the nearest approach both to the prevalence and intensity of the disorder at fixed times, afforded the means of measuring a standard of the disease by a standard of the meteorological states presented during the same times. I found, then, twelve weeks, viz., the weeks ending January 17th and April 4th, 1846; January the 2nd, April the 10th, June the 19th, July the 3rd and 31st, and August the 7th and 14th, 1847; and January 11th, February 1st, and March 15th, 1851; in all of which the mortality from scarlet fever was *sixteen* cases per week. I found five other weeks, viz., the weeks ending November the 2nd, 1850; September the 20th and 27th, and December the 20th, 1851; and January the 3rd, 1852; in each of which the mortality was *forty-one* per week. And finally, I selected two weeks, the one of extremely high, the other of extremely low mortality. The week ending October 14th, 1848, represented the first of these, for in this week there were *one hundred and eighty-eight* deaths; the week ending April the 18th, 1851, represented the second of these, there being only *eight* cases recorded.

Analysing these cases of death, and subjecting them to careful study by the side of the meteorological conditions, I constructed a table, of which the following is an abstract.

In the twelve weeks in which the mortality was sixteen per week, the *mean temperature* varied as much as 36°; one of the weeks having a mean of 65°, another of 29°. In the five weeks in which the deaths each

week were forty-one, the range of temperature was from 36° to 57°, giving a difference of 21°. In the week when the mortality was extremely high, viz., one hundred and eighty-eight, the temperature was 52°; whilst in that week in which the mortality was so low as eight, the mean temperature was 46°.

Turning from the thermometer to the *barometer*, I found an equal want of relationship. In the twelve weeks where there was an equal mortality per week of sixteen, there was a range in the barometrical readings varying from 30·277 to 29·435, or 0·842. In the weeks in which the mortality was forty-one, the reading varied from 30·190 to 29·625. In the week with the high mortality, one hundred and eighty-eight, the mean barometrical reading was 29·824; while in the week with the low mortality, eight, the reading was 29·776.

The Amount of Rain. In the twelve weeks of equal mortality of sixteen, the extremes were 1·61 to 0·00. In the five weeks in which the mortality was forty-one, the rain-fall was 0·15 to 0·00. In the week of the high mortality, one hundred and eighty-eight, the fall was 3·42; and in the week of the low mortality, eight, it was 0·02.

The Rate of Atmospheric Movement. In the weeks of equal mortality of sixteen per week, the difference was most varied; in one week there was absolute calm, in another the atmospheric movement had a mean of 207·857 miles per day. In the weeks with an equal mortality of forty-one, the range of atmospheric motion was from 428 to 975 miles per week.

In the week of very high mortality, one hundred and eighty-eight, the rate of motion of the air was 1,010 miles; and in the week of low mortality, eight, the movement was only 590 miles.

The Electrical Conditions of the Atmosphere showed a somewhat nearer approach towards one rule. In the twelve weeks, or eighty-four days, of equal mortality of sixteen, there were not less than fifty-seven days in which the electricity was *positive*; only three days in which there was negative electricity all the day; five days in which positive and negative electricity were shown on each day at different periods; and nineteen days in which the electrical results were nil. In the five weeks, or thirty-five days, in which the mortality was forty-one per week, there were three days and two hours of positive electricity; no evidence of negative electricity; but thirty-two days, minus two hours, in which no electrical results were given. In the week in which the mortality was one hundred and eighty-eight, positive electricity was developed on two days, negative on one day, and on four days none; while in the week of lowest mortality, eight, the electricity was positive on two days, and absent on five days.

From these records we can deduce, as I have already premised, but few facts of value bearing on the effects of special atmospherical changes on scarlet fever. It is worthy of remark, certainly, that during the period of eighty-four days when there was an equal weekly mortality of sixteen, there were fifty-seven days of positive electricity; while in the thirty-

five days, during which the mortality was at the rate of forty-one per week, there were thirty-two days in which there was no electrical manifestation. It might be inferred, hereupon, that the absence of electrical manifestation was connected with a high mortality, and, on the contrary, that the presence of positive electricity was connected with a low mortality. But when we see again a week of extreme high mortality, one hundred and eighty-eight, during which we have two days of positive electricity, and a week of extreme low mortality, eight, during which electricity was absent on five days, we can but accept the facts which seem to have an affirmative meaning in the light of mere coincidences. Yet it may be, that in the future some relationship between the presence of electricity and a low mortality from scarlet fever will be traced; for it is possible that, while electricity in no way interferes with the spread of the contagion of this disease, it produces modification in the effects of the poison on the animal organism.

But if there be only this one finger pointing affirmatively towards the relationship of meteorological conditions and scarlet fever, there is a large amount of negative evidence supplied in the statistics I have given. We see, for example, that, with equal mortality, the most opposite conditions of temperature may be presented; but the most interesting observation is that in reference to the movement of the air. Assuming that the disorder were propagated by means of a volatile poison, it is obvious that such poison is limited

in its range of action, or, at all events, that it is not influenced, by the mechanical vibrations of the atmospheric sea. In a dead calm the mortality may in one week be sixteen; and in another week, with a current of air passing over the infected spot at the rate of fourteen hundred and fifty-five miles per week, or two hundred and seven miles per day, the mortality shall be the same; while again with a week of extraordinary high mortality, one hundred and eighty-eight, a current of air may be sweeping over the home of the disease at the rate of one thousand and ten miles per week.

I know how many objections may be brought to bear on the method, laborious though it has been, by which the above considerations have been arrived at. It may be urged that mortality is not a sure indication of the prevalence of an epidemic; it may be urged that the meteorological conditions which attend the fatal end of a disorder are not necessarily the same as those which were present when the disorder was being communicated. And these objections are so valid that I would not dispute them. My sole object is carried out now, if I have indicated all that can be gathered from the best and only resources we at this present have at command.

The Recurrence of Scarlet Fever in the same Person is an interesting fact, and one that deserves to be made a subject of special study. On the possibility of such recurrence, opinion has been divided. The well-known assertion of Willan, that out of two thousand cases of the disease, he had never met with

one in which it had happened for the second time in the same person, has been transcribed by almost all writers, and has led many to entertain a belief that the disease could never occur twice in one individual. This belief, however, is quite erroneous. Rayer saw one well-marked instance of recurrence; Bateman and Withering saw several; Blackburne saw two such cases; and Dr. Tweedie observes—"We certainly have met with several well-authenticated instances of a second attack of scarlatina in the same person." I have known second attacks of scarlet fever in the same person without any doubt. A little girl whom I attended in 1850, took scarlet fever. The disease was very decided in character, but passed over favourably. A month afterwards, the little patient complained of feeling sick and cold, and I feared that the symptoms of dropsy were about to present themselves. Instead of this, the skin a second time became universally red, and the throat sore; the patient passed again through a most marked attack of scarlet fever.

I am able, too, to speak from experience in my own person on this subject, for I have suffered from the disease, not twice only, but thrice. When a child, I suffered severely from scarlet fever, during a time when it was occurring epidemically in my native village. The rash was universal, and intensely red; the throat sore; the recovery slow. In the spring of 1850, whilst attending cases of scarlet fever, I sickened, became ill, and passed through a very severe second attack of the disease. The skin was

again red, the throat sore and ulcerated, and the recovery gradual. Still more curious, whilst attending a boy, in the month of April 1852, who had a severe attack of scarlet fever, I became exceedingly unwell, suffered from shivering, and sore throat, and had a faint red blush on the chest and neck. I am minute in these particulars, because it is important to settle satisfactorily all points that admit of being settled in these inquiries; for by such means difficulties are exhausted, and fewer questions are left for investigation. I have not been able to collect statistical information on this subject of recurrences, so that it is impossible to state their frequency.

I take it, nevertheless, that the phenomenon of recurrence is most exceptional, and it is satisfactory to know that I can discover neither in literature, nor in general experience, one single case in which a second attack of scarlet fever has proved fatal.

I could not conclude this section on the natural history of scarlet fever, without reference to the *mortality of the disorder*, and specially in respect to the position which the disease holds in this particular to the other diseases of the zymotic class. Every writer on scarlet fever since its full recognition as a disease, has described, in mournful terms, its frightful ravages. Mr. Kearsley, of Philadelphia, writing about it a hundred years since as it appeared in America, says, "it baffled every effort to stop its progress, and seemed, by its dire effects, to be more like the sword of vengeance to stop the growth of the colonies, than the natural progress of a disease." To this day the yearly

victims of the disorder are exceedingly numerous in all parts of the world, wherever it exists.

In considering the mortality of scarlet fever, several questions force themselves on our notice, some of which have been already answered incidentally. I allude to the subjects of age, of sex, and of season.

Two questions still remain: first, *the influence of locality on the fatality* of scarlet fever; second, the *relative mortality* of scarlet fever in comparison with other epidemic diseases.

To examine these questions, I have had recourse to the pages of the Registrar-General, and have referred, therefore, only to the disease as it occurs in our own country. The table below shows the comparative mortality from scarlet fever, in towns and rural districts. To insure correctness, each statement of deaths was obtained from a population of a million.

Year.	District.	Towns.	Country.
1838.	London, 24 towns, and 12 counties	517	202
1838-9.	London and 5 counties . . .	2522	454
„	24 towns and 7 counties . .	1654	737
1841.	London, 24 towns, and 12 counties	683	684
	Mean from 1838 to 1841 . .	988	478

The mortality from scarlet fever in a million of people is, therefore, greater in towns than it is in rural districts.*

* While giving the above table as expressing the facts supplied, I think it fair to state, that later records show a less prominent mortality from scarlet fever in towns as compared with country districts. My

The following table indicates the different rates of mortality *per million* from scarlet fever, in eleven districts of England, during the years 1838, 1839, 1840, 1841, 1842, 1847, 1848. The last column will supply at a glance the comparative mortality.

District.	Deaths in seven years per million.	Relative order.
North Western Counties	10,328	1
Metropolis	7,838	2
Yorkshire	7,018	3
Monmouth and Wales	6,609	4
Northern Counties	6,428	5
North Midland Counties	6,244	6
Western Counties	5,013	7
Eastern Counties	4,708	8
South Midland Counties	4,694	9
South Eastern Counties	4,609	10
South Western Counties	4,122	11

The above table needs but few observations. It indicates an extraordinary mortality in the north-western counties, which still obtains. The fever does not fall, however, on the whole country at the same time, but is, at similar periods, absent to a great extent in one locality, and extensively present in another.

The subject of the relative mortality of scarlet fever in reference to other epidemic diseases, possesses peculiar interest; and I have taken some pains to illustrate it by several tables. The first of these shows the relative mortality of seven epidemic dis-

friend Dr. Greenhow, indeed, is of opinion that the mortality of towns is absolutely not greater: and that the annual mortality from scarlet fever in the healthiest country district in England is higher in proportion to the population than in either Liverpool, Birmingham, or London.

eases. The returns are, for England in the years 1838, 1839, 1840, 1841, and 1842, and for London in 1843. The calculations are based on the numbers of deaths in a population of a million, as in a previous table.

Diseases.	No. deaths.	Rel. order.
Typhus	6,442	1
Scarlet Fever	4,015	2
Hooping Cough	3,540	3
Measles	3,486	4
Small Pox	3,183	5
Erysipelas	488	6
Influenza	395	7
Total	21,549	

In special years, the relative position of these mortality figures is somewhat changed.

In the next table, the relative mortality in a total of 79,256 deaths from six epidemic diseases occurring in London during the period of twelve years, from 1840 to 1851, is exhibited:

Diseases.	No. deaths.	Rel. order.
Typhus	23,964	1
Scarlet Fever	21,551	2
Measles	15,460	3
Small Pox	10,710	4
Erysipelas	4,276	5
Influenza	3,295	6
Total	79,256	

In the following and final table the subject of relative mortality is carried further, by a calculation based on 462,227 deaths from seven epidemic diseases, occurring during a period of eleven years, viz., in England in 1838, 1839, 1840, 1841, 1842, 1847,

and 1848, in London in 1843, 1844, 1845, and 1846, and in Kent in 1843.

Diseases.	No. deaths.	Rel. order.
Typhus . . .	. 141,517	1
Scarlet Fever . .	. 105.250	2
Measles . . .	. 63,793	3
Hooping Cough . .	. 62,867	4
Small Pox . . .	. 59,460	5
Influenza . . .	. 18,553	6
Erysipelas . . .	. 10,787	7
Total .	. 462,227	

The figures thus quoted, place scarlet fever as second only in this country amongst the communicable diseases, in relation to mortality; nor does more recent investigation at all modify this relative order. On the contrary I believe that were the returns under the head "typhus" more correctly made, the disease scarlet fever would now stand highest on the list. It is unfortunate that the terms "typhus," and "typhoid," are used too frequently in a generic sense to chronicle asthenic pneumonia, uræmia, and various other maladies having a tendency in their later stages to assume what is called the "typhoid" type. There is, consequently, in all our records of disease, a preponderance given to typhus which, extensive as that disease is even now, it does not altogether deserve. It is so very important to see a rectification of this error in our statistical records, and the error is one to be so readily rectified by a little more attention to details of diagnosis on certificates of death, that I offer no apology for exposing it here, and for suggesting the means of its removal.

ON THE TYPES OF SCARLET FEVER.

It will be a good day when the three so-called distinctive forms of scarlet fever, viz., scarlatina simplex, anginosa, and maligna, shall be considered as one; for although it is strictly true that there are certain cases which, from the first to the last, adhere to one character, yet there appears to be no definite rule in respect to the conditions under which such definite forms are manifested.

I have kept notes these ten years of every fact that I have observed bearing on this subject, but without arriving at any absolute reason why differences of type should be maintained so persistently in different cases. True, I shall in a later part of this essay offer a possible reason for the typical forms; but in the present place I confine myself to the simple statement of observed facts. That there is no difference in the quality or quantity of poison in the respective types is clear; for I have three instances before me of fatal scarlatina maligna, ending in a few hours, in which the disease was contracted from communication with cases of the mildest kind. On the other side, I have evidence of examples in which exposure to malignant scarlet fever has excited the mildest type in other persons disposed to the malady. The unity of the poison is, moreover, further shown in the circumstance, that the frequent after effects of the acute disorder, viz., uræmia and dropsy, may occur after any type. This is the general rule; but I should add to it, nevertheless, that in so far as my experience teaches

me, acute uræmia following within a few days or hours on a scarlatinal attack, is most frequent when that attack has assumed the aspect of malignancy; while the prolonged anæmic albuminous dropsy succeeding to a scarlatinal attack is most common after one of the milder forms of the disorder. The experience of other observers may be contrary to mine in this respect; but although such difference may exist, the general fact is not shaken, that the same sequelæ may follow different phases of the acute disorder. It is also worthy of notice that the occurrence of scarlatina, in any of its forms, is equally protective; in other words, a patient who has suffered from simple scarlet fever is as well protected from the malignant type of the malady, as one who has already suffered from the malignant type. This fact is such a matter of common experience that it need not be dwelt on, except to enforce the identity of the poisons acting in both sets of cases.

That neither season nor peculiar known meteorological change is the cause of the distinction in type, is, I think, established by fact. It is true that sometimes a great epidemic of the disease breaks forth, and proves largely fatal, owing to the occurrence of numbers of cases, malignant in kind. But again, in these illustrations, if all the facts be gathered together, there are a proportionate number of mild, as of severe cases. It happens, not uncommonly, that a district of country remains for many years free from an epidemic visitation of scarlet fever. In the period, during which the disorder is absent, large numbers of

children are born, each one susceptible to the affection. Suddenly the disease is set up by the introduction of a case into the locality; and, the train once lighted, every house becomes the *habitat* of the dreaded visitor. In such epidemics as these, there are always a large number of malignant cases and a frightful mortality; but there is an equal number of milder cases always to be found, if sought for with care; and even a larger number of mild than of malignant cases, in proportion to the population attacked. My own observations would lead to the opinion, that the proportion of malignant to mild cases is as one to five; but this is a point which requires much more elaborate investigation than I have been able to bestow upon it; for, if there should be a law in the background regulating the proportion of malignant and benign cases in a given number of sufferers, the establishment of such law would give a new reading as to the cause of difference, such as has not before been surmised.

That the locality in which the disease occurs influences the type, seems to me equally unproven. I thought at one time, certainly, that in low dirty houses, the malignant tendency was most developed; but further observation has dispelled this view. For, although it must be conceded, that nothing more materially interferes with the successful treatment of the disorder, in all its forms, than the confinement of the patient in a foul unventilated chamber, I must confess to have seen the mildest phases of the complaint passed through in such places; while, on the

other hand, I have observed the severest forms of malignancy in the rooms of the well-provided classes, where the strictest cleanliness and the most perfect ventilation were provided.

Once more, the physical condition of the patient at the period of attack, seems as little to influence the course of his malady as the external conditions by which he is surrounded. I have known (and every other practitioner, I doubt not, has known the same) instances, in which the whole of a family of children having been attacked, the weakest of the members, the scape-goat of the whole, has passed through the stages of the fever with impunity, while the strongest have fought for every inch of life, or have actually succumbed. And this same observation holds good, to my mind, on a larger scale; for I can recall no fact to sustain the impression that the indifferently fed children of the poor are either more or less susceptible of scarlet fever than the well fed children of the rich, or are more or less susceptible of the malignant forms of the affection.

Lastly, there seems no evidence to support an hypothesis of hereditary tendency to any specific type. We see this hypothesis negatived in the simple circumstance that, in children born of the same parents, the disease may assume different types in different cases. I have also put the question as to the form in which parents of affected children did themselves suffer in early life; but the answer has never conveyed any such fact, as could show the sign of an hereditary claimancy to a specific variety. On the contrary, in

nearly every case that has ended fatally under my observation, the parents have assured me that they suffered from the affection in its mildest form. And yet I must add that, in one instance, where I had opportunity to investigate all the facts faithfully, a whole family of children seemed altogether insusceptible to the malady. A member of this family, on one occasion, slept with a relative during the time when she suffered from the disorder, and escaped. A servant was introduced into the house, and soon afterwards passed through the fever; she was tended by the family, and one of the circle, who had never suffered, though she was an attendant on the invalid, and, it is to be feared, conveyed the disease to another family, showed no sign of the disease herself. This insusceptibility appeared also to have been inherited from the parents of these children; the father and mother having, throughout their lives, escaped from the contagion, although their sisters and brothers had been sufferers from it.

Admitting, therefore, the view that hereditary predisposition to any specific form of scarlet fever does not exist, we may accept that, in certain very rare cases, there is manifested an entire insusceptibility of the disease, and that this insusceptibility is hereditary.

If, then, we allow (and I think we are bound to allow as much), that the variety of type of scarlet fever is not dependent on differences of poison, nor on meteorological state, locality, physical condition of the patient at the time of infection, age, nor on hereditary taint, where shall we look for an explana-

tion of the singular phenomenon, that of two children attacked at the same time, the one may be comparatively well at the period when the other lies dead, each event being encircled in the period of a few days? The question is one of the most profound and important in the field of medical inquiry. At this stage of our knowledge we must leave it with but one supposition; that there is, in the organism itself, a directing cause which modifies the violence of the poison, or intensifies its action; a cause allied to that agency which altogether prevents, in nearly every case, a repetition of the disease in those who have passed through its stages, whether gently or severely.

ON DOUBTFUL SCARLET FEVER.

I have twice met with examples of disease in which I have been at a loss to say, whether the cases were or were not scarlatinal in their nature.

A young woman, a servant at an inn, was seized with slight shiverings, sensations of chilliness, weight in the head, and thirst. These symptoms lasted about twelve hours, when the body became covered with a bright red rash, the throat sore, and the tongue loaded and white. She was kept in bed, and ordered a simple effervescing medicine. Twelve hours after the first appearance of the eruption, it had entirely disappeared, and the patient expressed herself as feeling quite well. Three weeks afterwards, the same symptoms returned, and passed off as favourably as before, leaving no secondary disease. The disease was communicated to no one else.

In the second case, the patient, a young married woman, was placed in less favourable circumstances; her room was imperfectly lighted and ventilated, her dwelling was situated in a dirty locality, and her attendants were indifferent nurses. A class of symptoms appeared, precisely analogous at first to those described above; but in this case, as the sore throat and eruption faded, great bodily prostration succeeded, attended with griping pains in the bowels and copious diarrhœa. This patient recovered very slowly, but communicated disease to none, although two children slept in the same bed with her, and an infant was at her breast.

The two cases thus described were in the same village (Barnes), and were occurring at the same time; but they were the only cases of the kind in the neighbourhood; and, although identical with cases of mild scarlet fever, so that no other name could possibly be given to them, they passed off without leaving any special sequelæ, and the patients propagated no disease to other persons around who were susceptible. The recollection of these cases has always left on my mind the fact, that there is another poison almost identical in its effects with scarlatinal poison, but which has not the physical properties rendering it capable of distribution and contagion.

ON SCARLET FEVER, AS COMPLICATED WITH ACUTE RHEUMATIC FEVER.

Under the term "dengue," or scarlatinal rheumatism, Dr. Wood of America, and a few other authors, have

described an epidemic, in which certain symptoms of scarlet fever are combined with those of acute rheumatism. Dr. Aitken, in his work on *The Practice of Medicine*, thus describes this epidemic:—"A peculiar febrile disease, conjoined with sudden severe pains in the small joints, which swell, succeeded by general heat of skin, intense pain in the eyeballs, and the appearance of cutaneous eruption on the third or fourth day. The disease is infectious, with an epidemic tendency."

After describing that the disease has been observed in certain parts of the East and West Indies, and in the southern parts of America, Dr. Aitken, adds—"*it*" (the disorder) *is not known in Great Britain.*"

Now, while it is true that we have no record of an epidemic of "dengue" in this country, it is certain that we not unfrequently meet with scarlet fever connected with acute rheumatic fever. I believe we were first indebted to Dr. Golding Bird for the mention of this fact. Afterwards, in 1845, at a meeting of the Belfast Medical Society, Dr. Kelso, of Lisburn, read a paper, in which he referred to twenty cases of the kind that occurred under his observation—(See report of proceedings of the Belfast Society in the *Dublin Hospital Gazette* for January 1st, 1846.) More recently, Dr. Ross has noticed very ably a similar conjunction of the scarlatinal and rheumatic disorders.

In April 1850, scarlet fever being then prevalent in the district, I attended a family on Barnes Green

that had been attacked with the prevailing disease. There were four children seized in this household; they were affected nearly at the same time. The introduction of the disease was clearly made out, and the fever took, in every case, the type of scarlatina anginosa. On the second day of the eruption two of the children, both boys, were seized with severe pains in the joints, particularly in the wrists, ankles, and knees, and on the following day there was as clear rheumatic swelling of these joints as in any case of acute rheumatic fever that I have ever witnessed; I saw, myself, at the same time, the scarlatinal skin and throat, the strawberry tongue, and the rheumatic joints. On the following day the rash had much subsided, and the skin, which had before been dry and hot, began to throw out the ordinary acid secretion of rheumatic fever. The next day the cases might be said to have assumed altogether the rheumatic state, and in one child the endocardial membrane became affected. For more than a week these two children appeared to be gradually recovering; but at the end of this period there was a recurrence of the acute rheumatic signs, together with indications of albuminuria. At this same time the two other children, who had passed simply through the scarlatinal disease, became affected with albuminuria, and also with pain and swelling of the joints. The recovery of each was extremely slow, owing chiefly to the frequent recurrence of rheumatic arthritis; but ultimately they did all recover, and that soundly. Singularly enough, during their

convalescence, an elder sister, who had not been affected with either scarlet fever or rheumatism, was seized with erysipelas of the face, and suffered dangerously from that disorder.

I could gain no clue whatever as to the cause of the admixture of the scarlatinal and rheumatismal symptoms in these children. The house in which they lived was opposite to a large mire or pool of water, but was not specially damp; nor were other children, situated near the same pool and also seized with scarlet fever, affected with joint-disorder. Further, I could not make out satisfactorily any proof of hereditary taint as accounting for the rheumatic complication; but there it was, and there was the fact, in spite of any hypothesis to the contrary, that two diseases may exist in the same body at the same time.

THE CHEMICAL PATHOLOGY OF SCARLET FEVER.

In considering the pathology of scarlet fever, it has been too common a practice to classify the disorder as belonging to fevers of the typhous series. Franz Simon enlarged this hypothesis much, by placing scarlet fever under the list of diseases marked by what he has termed hypinosis, or a condition in which there is less fibrine than in healthy blood, and in which the proportion of fibrine to the blood-corpuscles, 2.1 : 110, is less than is normal. He bases this classification of scarlet fever on four analyses by Andral and Gavarret; but it seems to me, even on the evidence of these analysts, that the dis-

order is falsely classified. In one of these cases the blood, at a first bleeding, gave an excess of fibrine, viz., 3.1 to 146 of blood-corpuscles; while at the second bleeding this proportion was increased, the fibrine being as 4.0 to 124.3 of blood-corpuscles. In their second case the fibrine was as 3.5 to 136.1 of blood-corpuscles, and in the third 6.8 to 112.2. Surely, then, it cannot be said that analysis of blood puts the disease out of the list of inflammatory disorders. I should urge that, so far as these very deficient inquiries go, they put the malady clearly under the inflammatory series, in which there is no decrease, but rather an increase, of fibrine—hyperinosis.

The pathology, as derived from the symptoms, can, moreover, only be read of as indicating the true inflammatory condition, in the original sense of that term; the circulation is accelerated, the free surfaces are unusually heated (I once saw the thermometer, with the bulb under the tongue, rise to 109° Fahr.), and in the throat and often in the external glands there are the *dolor*, *calor*, *rubor et tumor*. But if nothing else connected the disorder with ailments purely inflammatory in character, such as rheumatism and pneumonia, the condition of the urine would of itself sustain the analogy. In 1850 I observed that it was a frequent thing in scarlet fever to have, with the usually large increase of pigmentary matter, a free deposit of uric acid, as well as a copious deposit of deeply tinged urates. But more careful and longer continued observations have shown

that during the acute stage the urea is also increased, the water reduced, and the chlorine reduced; conditions almost identical with those which occur in pneumonia.*

That the amount of urine should be decreased in the early stages of scarlatina is, at first glance, remarkable, but is easily understood when another comparison is instituted, as between this disorder and pneumonia, or other diseases of the same class. For in them all, contrary to the general view, there is an excessive exudation from the skin. The position of this question was admirably put by Dr. Snow in his paper on the circulation in the capillaries. (Vide the *Medical Gazette* for 1842-3, volume 1, pp. 810-16.) In this paper, after quoting the experiments of MM. Breschet and Becquerel, which prove that the application of an impermeable gauze to the whole surface of the body of an animal stops transpiration and causes a gradual decrease of temperature, Snow proved that the same effects could be produced locally in his own person; since, by clothing his arm in an impermeable covering, he brought down the temperature in a marked degree. Moving from this fact to its opposite, Snow deduced with great acuteness, and with his natural precision he afterwards proved the deduction, that whenever the skin is hot and dry, during febrile states, the dryness is not an indication of suppressed, but of rapid excretion, due to the excessive emanation of caloric, and immediate evaporation.

* See Parkes, On the Urine, p. 263.

But if this view be applicable to all disorders of the inflammatory type, so is it most applicable in scarlet fever, where, with an universally heated state of skin, there are constant thirst, gratified largely by copious drinks, a decreased excretion of urine, and withal a decreasing weight of the body. With these general, there are, moreover, in scarlet fever local indications of the true inflammatory state. The bare fact that acute rheumatic arthritis is an occasional concomitant, is alone sufficient to prove the pathological position of the disease; for rheumatic inflammation could not coexist with anything less than an inflammatory diathesis: but more frequent examples of local inflammatory changes also mark definitely the same fact. Of these local inflammations the throat-affection comes first; next, I think, enlargement and suppuration of glands situated superficially. In one case, of which I have the notes before me, this glandular inflammation, commencing in the parotid and involving the whole side of the neck in what looked like an erysipelatous suppuration, did such destruction that the motor nerves of the face were destroyed, and the cheek was left permanently paralysed. Following after these results, purulent deposits in joints occur, perhaps, next in sequence; and after these, inflammatory products in the internal organs, especially in the lungs and spleen. I do not include in these indications of inflammatory conditions the kidney-disorder which is so common a sequela, because I am inclined to consider this rather as a mechanical derangement than an inflammatory result; but even

this point is one which remains *sub judice*, for exfoliation of epithelium is virtually little different from an inflammatory exudation.

Lastly, the mode of death during scarlet fever in the acute stage is essentially, in so far as my experience goes, the same as in all the true inflammatory affections. In this stage the blood, when drawn, is cupped or buffed; the blood-corpuscles show that well known attraction for each other which causes them rapidly to run together, and sink *en masse* to the lower part of the fluid; and the fibrine exhibits its tendency to slow and clear separation. And as out of the body the tendency is to a deposition of the fibrine, so in it the same tendency, commencing on the right side of the heart, leads to rapid death from arrest of the circulation. Even mild cases of scarlet fever sometimes rapidly, and without assignable reason to those who are unacquainted with the symptoms of fibrinous deposit, end fatally from this cause alone. I have written so fully in another volume on these forms of death, that I will not trouble the reader with the repetition of an old history in a new place.

THE POISON OF SCARLET FEVER IN RELATION TO ITS PROPAGATION AND ITS MODE OF ACTION.

The poison of scarlet fever, specific in its nature, is the most subtle of all the zymotic poisons. It travels on material objects with infinite ease, and may be laid up in material objects for any period; yet, left to itself, it seems incapable of transit to any considerable distance, the air alone not wafting it more than a few feet, if so far.

The poison, though minutely distributed, is almost certainly solid in respect to its own material property. If it were volatile, gaseous, it would be more dangerous than it is to those who surround patients suffering from the disease, and would rapidly lose all its dangers on distribution upon articles of clothing and the like. Being solid, however, it traverses over a limited area about the patient, but fixes on surrounding objects capable of receiving it, and abides by them until destroyed by heat or other disinfectant. Adhering in this manner to transmissible substances, the poison is carried in the most mysterious manner into the bodies of the susceptible. I know, for instance, of a well authenticated case in which the disease was conveyed into the family of a medical man by the mere transmission of a letter from the house of a friend many miles distant, in which one of the inmates was suffering from the disorder. The infected letter was unhappily seized by the child of the medical man, and was conveyed by her to her mamma. Both were susceptible, both contracted the disease, and both died of it.

For the same reason the poison can be laid up in the homes of infected families in the most singular way. During my early career, I assisted a practitioner at Saffron Walden in Essex for a considerable period. In our union district we had an outbreak of scarlet fever. At a short distance from one of our villages there was situated on a slight eminence a small clump of labourers' cottages, with the thatch peering down on the beds of the sleepers. A man and his wife lived

in one of these cottages, with four as lovely children as England ever owned. Not those immortal Angles whom Pope Gregory recognised as angels in the slave-mart of Rome were more worthy of our country. But the poison of scarlet fever entered this poor man's door, and at once struck down one of the flock. I had no time allowed me for the practice of any special remedy; but it seemed to me that I had saved the remaining children by obtaining their removal to the care of a grandparent, who lived at a village a few miles away. Some weeks elapsed, when one of these was allowed to return home. Within twenty-four hours it was seized with the disorder, and died with equal rapidity to the first. We were doubly cautious in respect to the return of the other children. Every inch of wall in the cottage was cleansed and lime-washed; every article of clothing and linen was washed, or, if bad, destroyed. Floors were thoroughly scoured, and so long a period as four months was allowed to elapse before any of the living children were brought home. Then one child was allowed to return, a boy nine years of age. He reached his father's cottage early in the morning: he seemed dull the next day; and at midnight in the succeeding twelve hours I was sent for, to find him also the subject of scarlet fever. The disease again assumed the malignant type, and this child died, despite all that could be done. I recommended after this event that the cottage should be newly roofed; but I am unable to say whether any such precaution was taken, for soon afterwards I left the

neighbourhood for good. I have always believed that in this instance the thatch was the medium in which the poison was retained.

The mode in which scarlet fever poison is received by the infected subject is, I think, unquestionably by the lungs. When in the year 1850 I contracted the disorder myself, I was conscious of the exact time, source, and place of the infection. I was called to a child in the Church Row in Barnes, who was stricken with the malady. It was one of the first cases I had visited in that epidemic. Knowing that I had once suffered from the malady, and having often attended other cases without experiencing any ill effects, I was quite sure of being safe, and therefore ran unlawful risks. The breathing of the child being hurried, and the alæ nasi working, I put my ear over the chest, without the stethoscope, a linen handkerchief alone intervening. I was conscious at the time of an odour from the patient, and of his warm breath playing on my face. I left the house; but I was soon apprised of derangement: I felt nauseated and chilly. I met Dr. Willis in his carriage on Barnes Terrace, and got him to take me home, telling him I was poisoned with the disease. If I had taken a dose of arsenic, the symptoms could not have been induced more methodically or inevitably; and it not a little relieved the anxiety which they brought me, to watch them in their course. The disease was very severe, but was followed by no serious consequences. The urine was never albuminous; but, if I am not mistaken, it is as rare for

the urine to be albuminous in adults after scarlet fever, as it is common in children.

In this example, the rapidity of the symptoms after the absorption of the poison gave direct evidence that the poison was taken in by respiration; for there was no abraded surface of skin for absorption, nor was any fluid imbibed between the period of exposure and the appearance of the symptoms. Moreover, I was conscious at the time of infection of the inhalation of a noxious air.

From what part of the body of the patient the poison comes, I am unable to decide; nor can I see any rational and easy method of experiment by which to arrive at a solution of the problem. Some inquiries made long since by one or two observers, as to the possibility of producing the disease by inoculation of the blood of the infected, would indicate, if anything at all, that there was no specific poison in the blood; while the probabilities are strongly in favour of the supposition that the poison is an excrete from the skin or bronchial mucous membrane. Whence, for example, in my own case, could I have received the poisonous agent, except from the breath or the skin of the patient? Yet it is to be observed, that it cannot be as gaseous matter that the poison escapes; for, if the poison were volatile, it would not remain in the clothing near to the body; or, inhaled by the recipient, it would not act in the same gradual way that it does; but, like all the volatile series, would produce an immediate impression, causing either rapid death or a state closely approaching death, but temporary

in its duration. All facts considered, I should be led to judge that the poison is thrown off with the epithelium or epidermis; or that it is itself simply epithelial or epidermic cells in a modified, *i. e.*, abnormal condition.

The mode of action of this poison in producing its effects on the economy is another point of deep interest. The consideration of this subject brings before us the whole question of zymosis, or animal fermentation. I have striven with considerable labour to understand the nature of this zymosis; and while the evidence is to my mind overwhelming in favour of the supposition, that on the entrance of certain organic products into the blood there is set up a change which gives rise to the symptoms, I am led to differ from other experimental authors as to the mode in which the process is conducted. The common assumption is that the organic poisons on their introduction into the blood undergo direct propagation and increase, and that upon their development and presence the specific symptoms of the disorder depend. Against this view I urge, that there is no evidence of the presence of anything approaching to a sporule, or cell, or organic germ, in the blood in any of the epidemic diseases: that many organic poisons, as the poison of the cobra, produce their effects too rapidly for the accomplishment of any organic cell-production: that fermentation out of the body is not necessarily connected with the formation of sporules or cells, but that these are accidentally derived: that if in the blood

there were constantly being formed these organic germs on the introduction of a single germ, there would never be recovery from a disease caused by such introduction, inasmuch as the development of new germs would continue so long as the body could supply, through the respiration and the circulation, the means of nourishment for the new growths: lastly, that the symptoms of the epidemic diseases can in many cases be closely imitated by the action of certain inorganic poisons with which we are well familiar. In the presence of these arguments, I for one am bound to throw aside the hypothesis of zymosis, whenever by that term is implied the idea of the production of specific cell-germs in the blood, or a dependence of symptoms on the presence of these assumed new developments. But on a simple chemical reading of zymosis, the theory may be accepted by the most sceptical, because it then becomes rational, and by analogy demonstrable, in its character. Let us thus consider the subject in its simplest positions.

Out of the body, the process of fermentation is excited when an albuminous product is brought into contact with bodies of the amylaceous or saccharine class; or, to formularise the production of the process after Liebig, "whenever decomposing albuminous substances are brought into contact with ternary compounds, such as sugar." The nature of the process seems to lie in the circumstance that the presence of the albuminous body gives to the oxygen the power to combine with the previously passive

elements of the ternary body. Thus, in alcoholic fermentation the sugar is transformed, if the decomposition be complete, into water, carbonic acid, and alcohol: while in the course of this process sporules or cells of the yeast-fungus are presented, the amount of these bearing proportion to the quantities of carbonic acid and of alcohol produced. But it is to be observed further, that this process may be modified, and that there may result lactic acid in lieu of alcohol—acid fermentation.

In the living body the zymotic process never, in so far as we know, passes to the point of the production of alcohol or of yeast sporule. For although the possibility of a blood fermentation yielding these products has been suggested, no demonstrative fact has been adduced, up to this time, in corroboration of the hypothesis.

But, as I have already tried to point out, in a paper on Zymosis published in the last volume of the *Transactions* of the Epidemiological Society, it is possible, and in some instances is demonstrable, that the development of acid bodies may occur in the human economy under zymotic influences. Some later researches lead me to doubt, however, whether the mode by which these products are produced, is described in that paper with the correctness I would wish at this time to supply. In constructing that paper, I was led by the common opinion to assume, that the transmissible poisons of the epidemic disorders act by setting up zymosis in the body as a new process. My more recent labours lead me to opine that, instead of zymosis being a process foreign

to the body, it is in truth a natural act, always going on, and essential to life. I derive from some experimental researches made within the last twelve months, that as the combination of oxygen with carbon and hydrogen in respiration is due to a decomposition of the respiratory parts of the blood, so the combination is brought about by the fibrine of the blood acting as a ferment; and thus that the evolution of heat is as much a consequence of this zymotic change, as it is a consequence in those changes which mark and identify the process of vinous fermentation.

On this view, then, the action of an organic body, entering from an infected organism into a healthy organism, is rather to modify or render imperfect the natural zymosis of the healthy organism, than to excite a primitive zymosis; and this view is supported almost to demonstration by the fact, that if to blood charged with amylaceous compounds oxygen be freely supplied, carbonic acid and water, with elevation of temperature, can be obtained as products: while if another body, in putrescency, which more or less destroys the combining power of the oxygen, be brought into contact with the blood, the result is that the process is either stopped altogether, or is so far arrested that the ordinary products, carbonic acid and water, are replaced by fixed acid products, of which the lactic acid is the type.

Whichever view be correct, the general principle is not implicated. Whether the poison thrown off by the infected subject excite in the healthy subject an

acid zymosis, or modifies the natural zymosis, the results are the same in regard to symptoms. The symptoms are caused, not by the propagation of vital germs, but by the development of a chemical body, which by its presence gives rise to the symptoms, and by its ultimate elimination frees the system of all the symptoms, except such as are secondary in their nature-sequelæ.

Thus, in regard to scarlet fever, as I take it, there are required for its demonstration—first, a susceptible blood; secondly, the introduction of a foreign body; and thirdly, a modified zymotic change, in which the ternary compounds are resolved into a soluble oxy-acid substance, which, passing into the systemic circuit, and brought to the skin and mucous surface as eliminating surfaces, acts there and everywhere as a direct irritant, and sets up those inflammatory changes by which the disease in its early stages is specifically characterised.

Of the nature of this secondary or systemic poison, the poison which is the direct cause of the symptoms, we have as yet no precise knowledge; but it is certain that it is to be found by a rigorous examination of the matters excreted by the subject of the fever; it is eliminated from the body, probably by the kidneys, perhaps by the skin, perhaps by the mucous membrane of the throat, perhaps by the bowels. So soon as it is found, the synthesis of scarlet fever will be a possible and an easy process.

From certain experiments which I have made for the production of acute inflammation of the serous

surfaces, from the existent analogies in symptoms between rheumatism and scarlet fever, and from the connection which may exist between the two maladies in the same case, I am, I feel, not far from the truth in suggesting that the two diseases, acute rheumatism and scarlet fever, are produced by similar poisons, and that the poison in each case is an acid having the physiological properties of lactic acid.

If we keep clearly in view the nature of the process given above, we are enabled (the explanation extends to other zymotic disorders) to see why it is the tendency of scarlet fever to present a series of acute symptoms, and then to pass away. The poisonous body introduced expends itself in the transformation of the fermentable matter of the blood into bodies of the acid series of fermentation-products; these products being capable of elimination from the system as they are formed, and the process by which they are formed stopping, the acute symptoms subside, leaving to be recovered from after effects which will be more or less severe, according to the amount of poison generated and the structural mischief which it shall have induced.

If this theory be true, it may explain the cause of the singular facts of non-recurrence of the disease, and of the decreasing tendency to it with increasing years. It is possible that in the young child the blood contains a substance capable of special fermentation, which in process of time, or under the eliminating action of an exciting poison, is removed from the economy. Nay, this same explanation may ex-

tend to the causation of the types of the disorder, the symptoms being mild in those in whom the fermentable substance is in small quantity, severe in those in whom the fermentable matter is present in a greater quantity.

In closing this section, I have for a moment to recur to the origin of the propagating poison of scarlet fever, the poison which passes from one subject to another. There can be no doubt that on the circumstance of every case of scarlet fever producing an identical poison, rests the common idea that this poison is the sustaining cause of the individual symptoms. I read the question in a different way. I look on the transmissible organic poison as the product of a modified chemistry in the affected subject, as excrementitious in its nature, and as a necessary antecedent to the symptoms, but not as the diffused irritant on which the symptoms immediately depend.

I believe, indeed, that organic chemistry will at some time be able to prove that the organic poisons, by which the various diseases of epidemic type are propagated, can be produced in the laboratory by a synthetical process of research conducted on blood; that these poisons will be put into our hands as substantive things, and labelled in our museums as prussic acid and the alkaloids are now presented. But this subject, so profoundly interesting to the whole of mankind, as constituting the very foundation of scientific medicine, I must reserve for a future work.

THE TREATMENT OF SCARLET FEVER.

I knew some time ago a gentleman, who for many years was proprietor of one of the largest schools for youths in the neighbourhood of the metropolis, who had a novel way of treating scarlet fever. The treatment may be called heroic, but it was heroically successful. Whenever any one of his pupils was seized with scarlet fever, he had the youth out into the playground, and, covering him well, walked him about until the perspiration streamed from every pore. This effected, the sufferer was taken indoors, put into a warm bed, charged with warm fluids, and made still to perspire. The patient usually fell into a gentle sleep, and in nearly every case was virtually cured at once. In many cases it would be impossible to carry out literally this plan of treatment; but in all cases the same purpose might be easily effected by the hot air bath. In either way, the essence of the treatment of scarlet fever in its early stages is effected, for the essence of treatment is to produce that which nature is aiming at, profuse excretion by the skin; in other words, to expedite as quickly as possible the expulsion of the products of a malchemistry from the blood.

In cases where it is practicable, the true hot air bath, so-called Turkish, would be used with most efficiency. Where this is not obtainable, the plan, which has long been in use in this country, of making the bed a warm air bath, may be resorted to with immense advantage. For this purpose a cradle

is placed over the patient, the bed-clothes are thrown over the cradle, and made to meet tightly round the patient's throat, so that he may breathe the outer air freely. A current of warm air is then driven by a long funnel-tube, made of tin, beneath the upper bed-clothes into the bed. Another very simple and convenient plan is to put a couple of heated bricks into a common warming pan, and to place the pan thus heated in the bed, at a little distance from the patient, keeping it in gentle motion to prevent any scorching of the clothes. By this means there is no risk of breathing carbonic acid, the head of the patient need not be specially excluded, and the cradle for raising the bedclothes may be dispensed with. For hospitals, a simply constructed hot air couch-bath is almost a necessary convenience.

Whatever means of this kind be employed, the object is the same—the production of a free diaphoresis; and this end achieved, the majority of the cases of scarlet fever would, in the presence of a pure air for respiratory purposes, recover naturally: but we must never forget that there is another element in the disorder, which may prove the fatal element, I mean the deposition of fibrine in the right cavities of the heart.

To meet this tendency to deposition, to prevent its occurrence, two remedies seem to me to be most reliable. The one is ammonia, as first recommended by Dr. Peart, and more recently by Dr. Witt; the other is acetic acid, as recommended by Mr. Isaac Baker Brown. I have often put these remedies into

use, in practice, and I am bound to say that they are both most effectual. It would be difficult to decide which is the best.

The action of these remedies at first sight may seem contradictory, but it is not so. Both acetic acid and ammonia have one property in common, that of holding the fibrine of the blood in solution. Both medicines also tend to eliminate carbonic acid; for ammonia, when freely administered, escapes from the skin, the breath, and perhaps the urine, in the form of carbonate; and acetic acid is decomposed entirely, escaping from the urine as carbonic acid in combination with a base. Further, by keeping the blood fluid, they favour elimination from every excreting surface. I have used with equally good effects a combination of these two remedies with an excess of ammonia, as in doses of two fluid-drachms of the liquor ammoniæ acetatis, with from three to five drops of liquor ammoniæ, in a liberal quantity of distilled water.

It is of importance in these exhibitions to administer the agent in small and frequently repeated doses, so that the blood may always contain the remedy; for, if the doses be given with intervals of three or four hours apart, the decomposition and elimination of the substance is over so quickly, that the system is left free of the effects during the greater part of the period between each dose. In a word, the secret of administration consists in putting aside the idea of a medicinal dose altogether, and in offering the remedy as a pleasant drink, rather than as a nauseous draught.

If the ammonia treatment be selected, there are two points particularly to be observed. The doses must be followed up until the agent is very distinctly presented in the breath. This presence may be determined by the holding of a glass rod, moistened with hydrochloric acid for the patient to exhale on; when, if much ammonia be expired, the white characteristic fumes will be developed. This test, however, is not very satisfactory. It is better to place a drop of pure hydrochloric acid on a microscope slide, and allow the patient to exhale some twenty or thirty times over the acid surface. Then, the glass slide being gently dried before the fire, or near a spirit-lamp, there will be left a crystalline deposit, which on microscopical examination will present the characters depicted in plate II, figure 1.

To effect this examination of the breath, Mr. Toogood, of Mount Street, has constructed, under my direction, a very simple pocket-tube, delineated in figure 2 of plate II.

The tube is in fact a strong test-tube, with a neck holding a stopper perforated in two places. One of the openings is armed with a mouth-piece, and the whole is covered in with a well fitted glass cap. When the tube is prepared for use, a microscope slide is moistened at one or two points with a minim of pure hydrochloric acid, and is inserted in the tube; the stopper and glass cap are then adjusted, and the whole may be carried in the pocket safely. When it is to be applied, the glass cap is removed, and the patient is made to expire gently

FIG. 1.

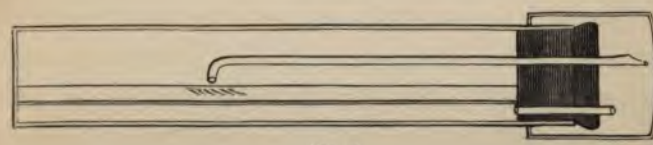


FIG. 2.

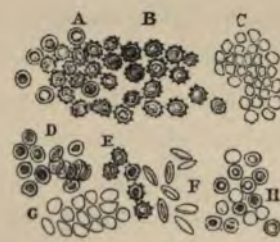


FIG. 3.

by the mouth through the mouth-piece some twenty or thirty times. The cap may then be replaced, and the tube removed altogether and examined at once, or retained for a time. Before microscopical examination the cap is removed, and the tube, the stopper still in it, is placed near a fire, or at a little distance from the flame of a spirit-lamp, in order to allow the chloride that may be formed to crystallise on the slide. When this is effected, the stopper is removed, and the slide is ready to be placed under the microscope for examination. The advantages of this tube are: 1st, that it is portable, 2nd, that in use nothing but the expired air of the patient comes into contact with the acid; and 3rd, that the evaporation and crystallisation are performed with as little exposure to the air as is possible.

A few experiments with this tube on healthy persons will indicate that, under most circumstances, crystals of chloride of ammonium are produced after expiration through the tube when the slide is charged with hydrochloric acid. But practice soon points out the extreme amount of the crystalline body that can be produced in health by a fixed number of expirations; so that comparisons between this production, and that occurring after the administration of ammonia, are easily and practically learned. In health, the crystalline deposit is thin and scattered; after free administration of ammonia, if the action of the skin or kidneys, or of both, be not profuse, the slide is frosted with the salt.

In administering ammonia, it is essential that the

medicine be pushed to the extent of showing a free evolution in the breath. But, while this point is to be insisted on, it is essential that there be a limit to the administration; for if it be carried to an extreme, two evils are induced. In the first place, the ammonia by its presence stops, to a considerable extent, the process of oxygenation; and, secondly, it acts by its solvent power on the blood-corpuscles. It is well, therefore, nay requisite, also to examine the blood microscopically during the administration; when, if the red corpuscles while still in motion present the appearances marked A, B, C, D, E, F, G, in figure 3, plate II—that is to say, if the discs be crenate, or irregular, or transparent, or collapsed, or oval, or very diffuse—the indications are given that the alkali is becoming dangerous, and requires to be suspended.

Whether the above methods of treatment (I mean the hot air bath and the administration of a blood-solvent) are the best and only means of treatment altogether, I cannot say. Time may come when an antidote immediate in its effects will be discovered, and which shall play to scarlet fever the same part as quinine plays to ague. But meantime, while the remedy is wanting that shall destroy the organic poison, or guard the body against it, the only two rational points of practice are the depuration of the secondary poison through the skin, and the administration of an alkaline solution having the properties of holding and retaining the blood in solution. We certainly address ourselves by these measures to the

effects of the secondary products of diseased functions, but our proceedings are none the more on that account to be ignored or despised.

If I have not named other plans of treatment, nor specified different and distinct treatments for differing types of the disorder, the omission must not be considered as accidental nor as inadvertent. The truth is that, in recasting the history of cases through three extensive epidemics, in which every variety and shade of treatment from bloodletting on the one side, to alcoholism on the other, was tried, I can extract no truth worth noticing as to treatment; except in those examples where, together with the induction of free elimination from the skin, and the administration of ammonia, fresh air and simple nutritious drinks, without stimulants, were supplied. In these examples, this treatment has seemed to me efficient; and I have now seen, in some twelve most severe instances, such unexpected results from it, that, without making out any pretence in it to a specific remedial system, I may speak of it as an approach towards a solution of the question of treatment, as it stands on the line of our present knowledge. I do not think cases need be considered in regard to their type for this treatment, but rather that, whenever treatment in any case is required, the general character of it is dictated in the outline I have given.

The local treatment, applied usually to the throat in scarlet fever, on which some practitioners exercise so much labour, is to my mind a secondary consideration. True, where there is much secretion, we

may avert asphyxia by removing the secretion by a sponge or piece of lint. But I am sure I have known great injury done by the excessive application of caustics; and, while I can recall no case in which the throat improved under any treatment while the general systemic mischief remained, I have never seen a throat, however disorganised, that did not recover when the general symptoms led the way to convalescence. There are, nevertheless, considerations which may lead to the employment of deodorising gargles or washes with much benefit. A weak solution of chloride of lime, or of chlorine water, is well adapted to such purpose; but the most elegant, as well as the most effectual deodorising gargle I have used, is made as follows:—

Of solution of peroxide of hydrogen, containing ten volumes of oxygen, six ounces; tincture of myrrh one ounce; rose water five ounces.

This gargle may be used *ad libitum:* it is very refreshing to the patient, and it removes the offensive secretions readily. In the case of young children who are unable to use a gargle, the throat is always easily washed by holding the little patient with the face downwards, and by pumping the solution over the surface of the fauces through a bit of gum-catheter, from a double acting India-rubber bag.

The preventive treatment of scarlet fever consists, first, in the carrying out of a rigid quarantine. Dr. Blackburn, half a century since, entirely proved that even in a boarding-school the disease would not spread, if the patients were confined to one room, and

the attendants were kept altogether away from the unaffected. Nor should the separation of the patient be confined merely to the time when the acute symptoms are being manifested; it should extend to at least a month after the date of the commencement of the attack. In the second place, the room of the patient should be kept sedulously ventilated, as well for his own safety, as for that of others. Lastly, as it is obvious that almost every material object, in contact with the patient, is capable of holding the poison, all articles of clothing and furniture should be thoroughly disinfected by the best of all disinfectants, heat at boiling point, before the susceptible body of another person approach them. With strict adherence to these simple rules, the disease, according to my experience of it, can be kept safely from propagation.

RESUME.

The propositions embodied in this contribution to the clinical history of scarlet fever are as follow:—

1. *Age.* Scarlet fever is most frequent during the third and fourth years of life. It declines rapidly after the fifth year, being nearly three times less common from five to ten, rather more than twelve times less from ten to twenty, nearly thirty times less from twenty to forty, and one hundred and one times less after forty, than in that period of life which is embraced in the first four years. The explanation of this fact lies, not in supposing that age, *per se*, exerts any decisive influence, but in the

circumstance that those who have not had the disease are susceptible to it, and that those who have had it are protected from it.

II. *Sex.* Sex exerts no influence, predisposing or other, on scarlet fever; male or female are alike infeeted, if they be alike exposed to the disease; so that any distinctions observed as between the sexes in a given population affected with the fever, are caused by difference in the numbers of the sexes present in the population and susceptible to the poison.

III. *Meteorological Influences.* The seasons materially modify the course and intensity of scarlet fever. The months of October, November, and December furnish in England the maximum amount of the disease; the months of April, May, and June the minimum; the proportions, without decimals, being as 9 for the months of April, May, and June, 11 for the months of July, August, and September, 14 for the months of October, November, and December, and 10 for the quarter included in January, February and March. The influence of season cannot, however, yet be traced to any special meteorological condition; neither temperature, barometric pressure, atmospheric movement, nor electrical modifications, can be shown as yet, to have any direct bearing on the prevalence or absence of the malady.

IV. *Recurrence.* Scarlet fever may recur once or even twice in the same person; but the event is comparatively rare, and death from a second attack is unknown as a fact.

v. *Mortality.* The mortality from scarlet fever is greater in towns than in rural districts; while, compared with that arising from other zymotic affections, it is statistically second only to that which attends typhus; and is probably, if the returns under the head "Typhus" were correctly made, first in relative order of the mortalities arising from the zymotici.

vi. *Types.* In the types of scarlet fever only one disease is recognisable. As causes producing varieties of type we may exclude differences of poison, meteorological conditions, localities, physical conditions of the patient, age, and hereditary predisposition. The only possible cause for difference of type which can be adduced, in the present state of knowledge, is, that in the susceptible organism itself there is a directing agency, which modifies the violence of the disease, or intensifies it. This view does not yet claim the rank of a theory.

vii. *Doubtful Scarlet Fever.* There is a form of disease, sporadic in kind, which in every symptom resembles scarlet fever. It lasts but a few hours, and does not seem dangerous. It may recur even within a few weeks after a previous attack. It is apparently not contagious.

viii. *Rheumatic Scarlet Fever.* Scarlet fever and acute rheumatism may occur simultaneously in the same person. The rheumatic symptoms may subside with or supersede the acute scarlatinal symptoms. The same rheumatic symptoms may recur with indications of albuminuria during apparent convalescence.

ix. *Chemical Pathology.* Scarlet fever belongs to

that form of disease manifestation in which the fibrine of the blood is increased. During the fever, the amount of urea and of uric acid excreted by the urine is increased, while that of the chlorides is decreased. Death commonly occurs from deposit of fibrine in the right side of the heart. Mild cases of the disease occasionally terminate fatally from this cause.

x. *The Primary Poison.* The primary poison of scarlet fever is probably solid in regard to its physical properties. It travels but a very little distance, except it be held in contact with some other body, such as an article of dress. It is destructible by heat at boiling point. It is thrown off from the affected person by the skin or the lungs, and is received by the susceptible person by respiration.

xi. *The Secondary Poison.* The symptoms of scarlet fever are probably due to the production in the economy of a secondary poison, having the physiological properties of an acid, such as the lactic. This secondary poison is the product of a modified zymotic process in the blood, induced by the absorption of the primary poison.

xii. *Treatment.* The curative treatment of scarlet fever consists in producing free action of the skin, and in maintaining the fluidity of the blood by the administration of a solvent remedy. The hygienic treatment consists in the admission of pure air to the patient, and in the establishment of a strict quarantine.

ESSAY V.

ON PULSATILE PULMONIC CREPITATION.

By "pulsatile pulmonic crepitation," I mean a stethoscopic sign, in which, without any indication of pneumonia or of tubercle, a crepitant sound, connected with pulsation, is presented to the ear. The sound was first described by myself at the Medical Society of London in 1854, and again at greater length in the *Medical Times and Gazette* for February 25th, 1860, under the title, "An Auscultatory Sound, produced by the Action of the Heart over a portion of Lung." At the time when this description was written, I had formed an opinion, that for the production of the sound the action of the *heart* on a tongue of lung was always required; I have since had reason to extend this view, as the sequel will show. I have learned, in a word, that other pulsating structures may excite the crepitation; and for this reason I have found it desirable to give to it a broader definition, and to denominate it "*Pulsatile Pulmonic Crepitation.*" Under this title the sound may now be classified as a stethoscopic phenomenon, having its own explanation and meaning as distinctly defined as ægophony, metallic

tinkling, or other recognised physical sign. To define the sound, I proceed at once to clinical records.

OBSERVED FACTS AND ANALYSES.

From the year 1851 to 1854, I was almost daily in attendance upon a lady, (Mrs. K.), who suffered from dilatation of the bronchial tubes and emphysema of the left lung: these conditions were attended with frequent attacks of urgent dyspnœa. In the last year of her life there appeared, without any acute premonitory symptoms, a well-marked systolic regurgitant murmur, evidently mitral, succeeded by general symptoms of dropsy. For some time, the abnormal heart-sounds continued without modification. The mitral murmur was fully pronounced, and was heard most distinctly towards the apex of the heart, the diastolic sound being perfectly normal and clear over the base. About six weeks before the death of this patient, after the subsidence of a very severe attack of dyspnœa, during which I was unable to make a physical examination of the chest, owing to the oppression which the act produced, I was surprised, when the examination could again be borne, at hearing over the cardiac region an auscultatory sound, which was not only new to this special case, but new to my ear altogether. The systolic rasp was present, as before; it was most distinct about three inches below the shrunken nipple, bearing a little to the left side. The diastolic sound was still perfect; it was most distinctly heard about an inch in a straight line upwards above the left nipple. But an inch below the nipple,

and bearing to the left side, there was a superficial sound which could be localised by the mouth of the stethoscope, and which, when present, obscured the systolic rasp altogether at that single point of observation. The sound was so superficial, that it seemed directly beneath the stethoscope; or, if the ear were applied to the chest without the stethoscope, immediately beneath the ear. It was not a friction-sound, it was not a murmur, it was not an ordinary crepitation; it was rather a coarse crackling noise, resembling that which is produced by the burning of dried gorse, or the tearing of a piece of calico. The period of the sound was also peculiar: it was irregular as to time; and sometimes, with the stethoscope over the spot where it was most marked, it would be absent during one entire action of the heart, or even longer, during which period of absence the systolic murmur and the clear diastolic *dup* were distinctly rendered. This observation of an occasional absence led me ultimately to the cause of the sound. When the sound did appear, it was synchronous with the systolic rasp, and clouded it; the second sound followed unchanged. After a little inquiry, I found that the new sound was in some measure influenced by respiration; this influence resolved itself into the following facts. When the patient expired, and forcibly opposed the refilling of the chest with air, the new cardiac sound was absent altogether. When the patient made a deep inspiration and held the breath, the sound was produced, so long as the inspiration was sustained, at every systole of the heart. At the beginning of an

ordinary inspiration the sound became elicited; at the acme of inspiration it was most marked; towards the close of expiration it was feeble, or lost altogether. To distinguish these differences, a slow and full respiration was necessary. When the respiration was quick and feeble, the new sound was always presented at the systole, so that it seemed then to be exclusively connected with the heart. The late Dr. Snow saw this case with me, and on two occasions confirmed all the facts I have named above.

A second example of pulsatile crepitation occurred to me in a case which I attended with Mr. Gaskell, of Chelsea. The patient, a young man, twenty-one years of age, the son of a well-known solicitor, after an acute attack of inflammatory fever, which assumed a remittent rather than a rheumatic type, but during which the endocardium became implicated, suffered severely from the effects of chronic endocardial mischief. When I saw him three months before his death, I diagnosed mitral and aortic disease, with hypertrophy of the heart, and tubercle of the left lung. I saw him weekly, Mr. Gaskell attending in the intervals. During one of these intervals, our patient had an acute attack of pleuritic pain in the left side, which lasted four days, and embarrassed the breathing very considerably. On examining the chest at my next visit, I was surprised to find an exocardial sound, identical in character with that I have described as present in the previous case. The sound was at the base of the heart and to the left side. As before, it was super-

ficial, local, an accompaniment of the systole, painfully distinct, and subject to similar modifications, as in the last named case, during inspiration and expiration. Mr. Gaskell and I often listened to this sound for many minutes together. I put an iodine mark round the spot externally, where the sound was best heard; the ring, not larger than a crown-piece, localised the position of the sound until the death of the patient.

I met with a third case in which the same sound was presented, in a child who came before me at the Royal Infirmary for Diseases of the Chest. This child was the subject of the chronic effects of hooping-cough. There were distinct physical indications of enlarged bronchi, emphysema of the right lung, and hypertrophy of the heart. The ordinary systolic and diastolic sounds were, in this instance, unattended by murmur. The exocardial sound existed again at the base of the heart, and bearing to the left side. It was coincident with the systole, and was also modified by the respiration, being prevented by a long expiration and brought out by inspiration; but, as the child could not be taught to inspire and expire by rule, the facts were not always well defined. Unfortunately, I am unable to follow out the history of this case further. The child either died, or was taken for treatment to some other Institution. She was brought to the Infirmary but once.

The above three cases were published in my pre-

vious essay; but since then I have met with an instance in which the pulsatile crepitation was detected at another portion of the thorax. A private patient, who called on me one morning from the north of England, complained of symptoms which indicated the presence of phthisis. He was a tall, thin, anæmic young man, who had been much confined in an office, at the desk. He had suffered slightly from hæmoptysis, had night-sweats, and was losing flesh and strength. The right lung, at the apex, rendered unmistakable evidence of tubercular deposition in an early stage. On the left side there were, here and there, bronchial cooings; and the posterior part of this lung had been the seat of sharp pleuritic pains. On placing the stethoscope to the left of the vertebral column, and very low down—at the base, in fact, of the left lung at its posterior and inner margin—I caught a pulsatile crepitation, much finer than that which had been observed in the preceding cases, but unquestionably governed by the same influences. When a long expiration was made, the crepitation was lost; when a long inspiration was made, it reappeared. This might have indicated nothing more than crepitation from tubercle; but the sound happened only in conjunction with the action of the heart, being coincident with the systole, synchronous with the pulse, and as perfect in its occurrence as the pulse itself. It was obviously connected, therefore, with *aortic* pulsation.

Analysing the auscultatory symptom thus described, we are led to trace it to a point connecting it with the acts both of respiration and of circulation. It is pro-

duced only in the precincts of a pulsating structure, such as the heart or a large artery; and it is obviously brought out by the action of the pulsating organ. In so far, then, it is a circulatory sound. But, again, it is not capable of being elicited except in the chest, nor there unless the lungs be distended with air; the air in the lungs may be held in the statical condition, but it must be present. Hence the sound is strictly compounded of two acts; one, inspiratory or predisposing; another, pulsatory and exciting.

The cases in which the sound is met with are all of a kind in which there are signs of thoracic disease. In two of the cases cited, there were extensive cardiac lesions; in one there was mitral murmur, and evidence of enlargement of the whole heart: in the other, the physical indications pointed out the existence of mitral and aortic disease, also with great enlargement. In the remaining cases, the circulatory apparatus was apparently healthy. In all the cases there was marked pulmonic disease, which offered one analogous fact throughout, viz., unmistakable signs of pre-existent pleurisy; two of the patients had also been sufferers from long standing bronchial disorder and emphysema; the other two gave evidence of tubercle.

CAUSE AND CAUSATION OF PULSATILE PULMONIC CREPITATION.

The study of these facts of diagnosis, taken in connection with the character of the pulsatile crepitation itself, led me early to the cause of the sound. It occurred

to me in the case of Mrs. K., that a portion of the left lung, at the point where it partially envelopes the left side of the heart, had been drawn downwards by pleuritic adhesion, and bound to the thoracic wall immediately over the heart; that the piece of lung thus placed was subjected to the impulse of the heart during systole; and that, when the lung was inflated on inspiration, the sudden compression, produced by the impulse, elicited the crepitation by forcible expulsion of air from the air-vesicles. I explained this view to Dr. Snow, who concurred with me entirely. In the succeeding two cases the same explanation presented itself; in the last case a like exposition was suggested, except that in this example the aorta, instead of the heart, was the pulsating body. Let me now show how far this diagnosis was proved correct.

Post mortem Facts. The first two cases above recorded ended fatally, and both, curiously enough, exactly six weeks after the occurrence of the pulsatile crepitation. I look upon this circumstance, of course, only as coincidental. The first patient died slowly, from exhaustion. The second died while he was sitting up in bed, laughing at some passing event.

The *post mortem* examination, in the first case, revealed no disease of the segments of the mitral valve itself; but in the left ventricle, immediately below the valve, and to the left side, there was firmly attached to the endocardial membrane a rounded concretion, the base of which was of the size of a shilling, and the projection of which into the ventricle

extended for at least half an inch. The concretion was partly organised. The heart was hypertrophied. The pericardium was adherent to the lung, and over the base of the left ventricle, to the left side, in the exact position over which the pulsatile crepitation had been heard during life, there extended a corner or tongue of lung. This structure, bound down to the pericardium by the under surface, was attached to the thoracic wall above by a band of adhesion an inch broad, which was inserted, almost like a fine tendon, into the thoracic wall close to the sternum. This band pulled the lung quite over the heart in an oblique direction. On its under surface the piece of lung was compressed into a concavity, into which the rounded surface of the heart fitted; the appearance of the lung indicated that the structure had been subjected to compression at this overlapping point. The parts as thus presented were shown at the Medical Society of London in 1854.

In the second case, extensive disease was found on the left side of the heart. The segments of the mitral valve were thickened and indurated; on the aortic valves were atheromatous deposits; and there was an extension of atheroma into the aorta. The heart was unusually large from dilatation, with parietal hypertrophy.

At the base of the heart externally there was the same condition as was observed in the preceding example, except that there was no adhesion of the lung to the pericardium; but a piece of the left lung, at its lower anterior margin, two inches in length and

tongue-shaped, was dragged obliquely from above downwards over the heart by a membranous band. The band was firmly attached to the sternum by its extreme end. The portion of lung passing over the heart was compressed on the under surface into a concavity, and its structure was condensed. The centre of the compressed spot corresponded above with the centre of the ring which I had marked on the thorax externally by the iodine line.

To make the fact of causation more evident still, I performed, in this inspection, the following experiment. I placed a piece of tubing in the trachea, the lungs and heart being still *in situ*, and made my assistant inflate the lungs with air, whilst I, seizing the extreme point of the tongue of lung, extended it in the line in which it had been bound; then, when the whole lung was full of air, I forcibly compressed the tongue of lung between the fingers and thumb of my free hand, and, holding the stethoscope between the back of the compressing hand and my ear, I elicited, with each compression of the hand, the same sound precisely as I had heard during the life of the patient.

OBSERVATIONS BY DR. WOILLEZ.

At the time when I first described the case of Mrs. K., in 1854, the sound which is here designated as "pulsatile pulmonic crepitation" had not, I believe, been noticed by any other observer; and, indeed, the same remark applies to the paper published in the *Medical Times and Gazette* in February, 1860. But I

observe in *L'Union Médicale* for June 16th, 1860, that Dr. Woillez has reported to the Société Médicale des Hôpitaux the particulars of a case in which the same sound was detected. Dr. Woillez's case occurred in the person of a young man, aged 22, a medical student at Vienna, who had a peculiar thoracic deformity. This patient, though delicate, had experienced no inconvenience in breathing, nor pulmonary disease. There was also in him no hereditary predisposition to thoracic disorder. In the anterior part of his chest there was an irregularly oval depression, funnel-shaped, nearly ten inches long, in the vertical direction, *i. e.* from the commencement of the cleft of the sternum and internal portion of the clavicles as far down as the level of the epigastrium; seven inches in the transverse direction; and two inches and three quarters deep. Apart from this deformity, the general conformation was satisfactory; there was no lateral deviation of the vertebral column. In a word, there was simply extreme depression of the sternum.

The physical signs presented by Dr. Woillez's patient were peculiar. On percussion, there was a slightly dull sound at the level of the centre of the heart. On auscultation, the maximum of the impulse and of the sounds of the heart was distinctly perceptible at the level of the cartilage of the third left rib, and these sounds were normal. At the apex the sounds were obscured by a portion of lung, the vesicular murmur being here discernible. The respiratory movements were mainly by the upper ribs. The

respiratory sounds were weak in the two sides of the thorax, but tolerably strong in the axillary region on the right side. On the left side of the thorax posteriorly, the beatings of the aorta were distinct. These beatings were heard in the upper part of the back, immediately to the left of the spine; but diverged from it in proceeding downwards to a distance of about two inches and a half, opposite to the deepest point of the sternal depression. At this point a sound was distinguishable, very different from the simple arterial sounds perceptible above. The arterial sound heard at the level stated, *was not uniform nor well defined: if the patient were told to hold his breath, an intermitting vascular sound was heard, regular and synchronous with the pulse, and which was neither a blowing sound, nor a rasping sound, nor a simple arterial bruissement, but a sound manifestly granulous in character, beginning and ceasing with the beating of the artery, but without an analogue in the anomalous vascular sounds which have been described. Moreover, when the patient was made to resume his breathing, immediately the normal vesicular murmur and the aortic sound were heard again. Each new examination, often repeated, produced identical results.*

Apropos of the aorta, Dr. Woillez explains the cause of the "granulous murmur" as follows: "The aorta, being several centimètres distant on the outside of the vertebral column, was necessarily surrounded on all parts except behind by pulmonary tissue; for the respiratory murmur was heard to the inner as well as to the outer side of the aorta. At the level of the maximum of the anterior sternal depression there-

fore, the contiguity of the lung and of the aorta must have been very close, whence the rubbing of the pulmonary tissue at each aortic dilatation gave a granular sound, as if the air were driven successively from several pulmonary cells." This explanation of the sound heard by Dr. Woillez is the same as that which I had already given in the cases reported above. I name this fact specially, because, from a note which he has made on those cases, he seems to have misinterpreted my reading of the phenomenon, and to convey a difference of opinion between us which in reality does not exist.

DIAGNOSIS OF PULSATILE PULMONIC CREPITATION, DIFFERENTIAL AND ABSOLUTE.

Differential Diagnosis. There are four physical signs of disease, diagnosable by the stethoscope and by percussion, with which pulsatile crepitation may be confused. These are:—

PNEUMONIC CREPITATION.
TUBERCULAR CREPITATION.
MOIST FRICTIONAL CREPITATION.
MINUTE MUCOUS *Râle.*

Pneumonic Crepitation, at first sight, and on careless analysis of facts, may easily be confounded with pulsatile crepitation; for it usually happens, that this last-named specific sound is heralded by an acute inflammatory paroxysm, in which there is much thoracic and febrile disturbance. The distinction in diagnosis rests on two broad differences. 1st, the pulsatile crepitation is louder and coarser than ever occurs in pneu-

monia. 2nd, The crepitation is not absolutely governed by respiration: it is not absent when the breath is held, but is then most fully developed; it is presented in alternation with inspiration and expiration, during ordinary breathing, but is always synchronous with the beat of the heart or the pulse. 3rd, It is localised to particular points; *i. e.*, to points where a portion of lung, a pulsating organ, and an opposing thoracic wall, are all brought into one focus.

Tubercular Crepitation may be co-existent with pulsatile pulmonic crepitation, in the same case. But tubercular crepitation may be known by the ear directly as a different phenomenon. If the tubercular sound be that which indicates an early stage of tuberculosis, its fine pneumonic character is sufficiently differential. If the tubercular crepitation indicate a late stage of tuberculosis, the moist character of the crepitation is equally differential: for the pulsatile crepitation, though loud and coarse, is essentially *dry*. Add to this, that the tubercular crepitation is independent of any pulsation, and is governed purely by the respiratory acts, and the distinction between the two phenomena is sufficiently clear.

Moist Frictional Crepitation, occurring in cases where, with tubercular deposit situated on the outer surface of the lung, there is slight pleuritic effusion, might be mistaken for pulsatile crepitation by the unwary; for in these cases the abnormal sound is superficial. But the crepitation in these tubercular pleurisies is moist and small, and is brought out only by a respiratory act. I have seen two cases where,

with tubercle in the lower anterior margin of the left lung, there was moist pleuritic friction; and, the impulse of the heart being plainly perceptible over the same region, a certain analogy was detectible, as between the sound thus induced and the pulsatile sound. But, on careful inquiry in both patients, the fineness and moistness of the crepitation, and its occurrence independently of the cardiac rhythm, were so obvious that no doubt could remain as to the diagnosis.

Minute Mucous Râle, occurring from effusion into the finer bronchial ramifications, is the last sound with which I am conversant, as possibly mistakeable with pulsatile crepitation. But every one who can distinguish a râle from a crepitation, can distinguish the difference required for this diagnosis; not to say, further, that the râle is synchronous with respiration and independent of the circulation,—not pulsatile.

Add, lastly, to all these facts, this one: that whereas, in cases of pneumonic crepitation, acute tubercular crepitation, moist frictional crepitation, or minute mucous râle, there is dulness on percussion over the seat of the auscultatory sound, while there is resonance on percussion over the seat of pulsatile crepitation; and the differential diagnosis is as perfect as can be desired.

Absolute Diagnosis. Pulsatile pulmonic crepitation is a loud harsh crackling sound, resembling that which may be produced by inflating a piece of lung-tissue, and, during the inflation, making forcible quick compression of the lung, so as to dislodge rapidly the air from the vesicles. The

sound is heard only at the time when a pulsating structure, near to the point where it is met with, is presenting its impulse. Even then, it is elicited only when the lungs are charged with air. Sustained inspiration gives a condition in which the crepitation is presented at every impulse of the pulsating organ. Sustained expiration removes the crepitation, leaving to be heard the simple pulsatile sound of the vascular structure, and nothing more. The sound may be met with over the heart, *i.e.*, between the heart and the thoracic wall; or over the aorta, *i. e.*, between the aorta and the thoracic wall. Percussion over the points where the crepitation is detected yields a resonance, more or less clear according to the state of the respiration. During inspiration the resonance is very clear, during expiration it is reduced, but is less dull than is common on percussion over the body of the heart. Lastly, the sound under the stethoscope is superficial in character, seeming as though it proceeded from a limited point immediately below the thoracic parietes.

BEARINGS OF PULSATILE PULMONIC CREPITATION ON PRACTICAL MEDICINE.

In the matter of treatment, a knowledge of the existence of pulsatile crepitation is of no affirmative value, or at most of little. The condition of lung which disposes to the sound is in itself comparatively harmless; and were it not so, it were difficult to devise any safe remedial means for removing the condition. But negatively, a knowledge of the nature

of the sound may prevent any mistake respecting its connection with pneumonic or other acute local inflammatory states, and may also prevent the employment of useless measures, which the hypothesis of the existence of inflammation as its cause might suggest.

In respect to prognosis, the sound has in some cases a definite meaning; in cases for example, where, as in the fourth case narrated by myself, there is, with tubercular crepitation anteriorly, pulsatile crepitation over the aorta behind. In such cases the explanation of the crepitation, without the necessity of assuming a posterior deposition of tubercle, throws a favourable cast over the prognosis; while in such an instance as that recorded by Dr. Woillez, the true reading of the cause of the crepitation removes from the mind of the practitioner all apprehension of danger, and converts what might in ignorance be considered a formidable sign into an interesting and harmless phenomenon.

The main value of a knowledge of pulsatile pulmonic crepitation relates, however, to diagnosis. For my own part, well drilled and taught in the science and art of physical exploration, I was for a period of several days utterly unable to explain the new sound, after first becoming acquainted with it in the case of Mrs. K. Indeed, my first impressions led me to attach much more importance to it than it really deserved. It occurred to me, in the first place, that the sound was possibly due to the fluctuation of some fluid enclosed in a cyst attached to the pericardium or to the outer surface of the heart, and

communicating, for aught I knew, with the inner surface. Next, I thought that it might occur from some deposit of calcareous substance on the pericardial surface; and, in fact, I was embarrassed as much with doubts and fears, as any practitioner, in an imminent and doubtful case, can well be. At last, the true explanation of the phenomenon presenting itself, these anxieties were all cleared away; and now the diagnosis of this special auscultatory sound is to my ear as definite and as simple as that of any much more commonly met with. To make others equally and readily conversant with the sound is the simple object of this essay; and as the phenomenon described is one, the verbal account of which is almost sufficient to lead to its immediate recognition, I hope I have succeeded in rendering it familiar even to the novice in auscultation.

ESSAY VI.

ON URÆMIC COMA.

THERE is no one class of symptoms grouped together in our modern nosologies under a specific name, of greater or wider import than that which we are accustomed to recognise under the term *uræmia*. Bearing in many instances a close analogy to other symptoms produced by known narcotic poisons, as well as to the symptoms of apoplexy and even of epilepsy, the pure indications of uræmic coma cannot be too early learned by the student, nor too carefully studied and recast by the matured practitioner. In the present essay, it is my object to speak only of the uræmic condition as it absolutely presents itself in its most marked representation. As the patient appears, when, comatose and convulsed, he lies the prostrated victim of uræmic toxæmia, I write of him; of his symptoms, of his pathology, of the immediate cause of his malady, and of the treatment which promises most certain and effectual relief.

OBSERVED FACTS AND ANALYSES.

A man, sixty-five years of age, who had been many years a letter-carrier, was seized on November

3rd, 1851, with drowsiness whilst delivering his evening letters. He went home and to bed, where he fell into a profound coma, preceded, however, by severe pain over the right hypochondrium. He had for some time before suffered from constipation, and had been passing albuminous urine. On the morning of the 4th, he was seen by a medical friend of mine, who found him typhoid and comatose: he sank rapidly, and died at half-past seven in the evening of that day.

The friends of the patient being of opinion that he had taken, either on his own account or by maladministration, some narcotic poison, I was retained to make a *post mortem* examination. This was conducted fourteen hours after death.

The body was still warm; the limbs were flaccid. On opening the chest, I found some slight old-standing adhesions between the visceral and the parietal pleural membranes on the right side. In the apex of the right lung were two small cavities; but there were no signs of tubercular deposit. The heart was loaded with fat externally, although there was great emaciation in all other parts of the body, and hardly a trace of adipose deposit. The heart was soft in structure, but not fatty. The right ventricle contained a soft fibrinous coagulum, which sent ramifications into the pulmonary artery. The liver was healthy. The spleen was shrunken, and very firm in its structure. The small intestines were normal, except at the lower part; here, for a space six inches in length, the ileum was reddened and injected on the

inner surface: the mucous surface of the cæcum was also much injected, and the surface of the ascending and transverse colon was in the same condition. There was no obstruction anywhere in the canal, and the tube contained nothing except mucus. There was no ulceration of the intestinal glands. The kidneys were disorganised; the pelvis of each was filled with a soft fat; the organs were large, soft, and white; the tubular structure was entirely degenerated, and the cortical soft and greasy to the fingers. The other parts of the body were natural. I could find no indication of poison, mineral or vegetable, in any of the tissues.

The succeeding history is more interesting in that, owing to the circumstances by which it was surrounded, it led to a coroner's inquest, and to an important medico-legal investigation.

A woman, aged thirty-four years, who was given to the free use of alcoholics, and had recently been treated for primary syphilis, was seized on the 18th November, 1859, with rigors. She was attended by a neighbouring chemist, who on November 22nd gave her a mixture, which he said contained quinine, dilute nitric acid, nitrate of potassa, syrup of buckthorn, sulphate of magnesia, and water.* I proved afterwards that these were

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* The formula supplied was as follows.

℞ Quinæ disulphatis gr. vj; acidi nitrici dilut. ʒ iss; potassæ nitratis ʒ ss; solutionis magnesiæ sulphatis ℥j; syrupi rhamni cathartici ʒ iij; aquæ ad ℥vj.

Two tablespoonfuls to be taken every four hours.

the constituent parts of a remaining portion of the mixture. The medicine was sent in to the woman on the evening of the 22nd; and, after taking a large quantity of beef-tea, she swallowed one dose of the mixture. Five minutes after this event she became hysterical and convulsed, and the friends, believing the woman to have been poisoned, called in the druggist, who in alarm tried to get her to take some ipecacuanha, but without avail. In the course of the night, Mr. Harris, of Windmill Street, was summoned: he found the woman in a state of typhoid coma, with the pupils slightly dilated and immovable, and the body at times convulsed, the convulsions assuming an epileptiform type. Mr. Harris called to his assistance my friend Mr. Peter Marshall, of Bedford Square: they carried out every available means of treatment; but the coma became more profound, and, seventy hours after the administration of the mixture already named, death closed the scene. By the coroner's warrant the body was examined, and a chemical inquiry instituted. Mr. Marshall, Mr. Harris, and myself were engaged in this research: Mr. Callender was present to watch the case on the part of the accused druggist, together with Mr. Morgan.

The autopsy was conducted thirty hours after death. There was no cadaveric rigidity at that time; there were no marks of external violence; there were evidences of the specific disease under which the patient had suffered, viz. warty excrescences on the pudenda.

There were *post mortem* ecchymoses on the back, and marks of a recent blister at the nape of the neck.

The body was very plump, and filled out with adipose deposit. There were no evidences of congestion of the brain, nor of effusion; the membranes were natural. The ventricles contained no fluid. The structure of the brain was normal, except, perhaps, that the thalami optici were slightly softer and more red than is common: there was a difference of opinion on this point; the medulla, the pons Varolii, and the cerebellum, were all quite healthy.

In the sinuses were found separations of fibrine. There was a small separation in the straight sinus; and the lateral sinuses, filled in their posterior third with fluid blood, contained in their remaining course fibrinous casts.

There was no evidence of thoracic effusion; but the pericardial cavity contained half an ounce of serum, which on exposure to the air did not coagulate. The pericardial membrane itself was quite healthy. The heart was pale, with a greenish tinge derived from commencing decomposition; on its outer surface it was largely loaded with fat; there was no congestion of the external vessels; the right cavities contained very frothy fluid blood. In the right auricle there was a dark gelatinous clot, which, extending into the ventricle, sent prolongations into the pulmonary artery; this clot was soft, and, when it was cut into, serum exuded from it. The walls of the right ventricle were attenuated to the last degree; they were not thicker in parts than cartridge-paper, and, when held up to the light, showed points where a faint transparence was discernible; the structures

throughout were deeply stained with blood; the ventricular and semilunar valves were quite natural. The walls of the left ventricle were thin and pale; the surface cut greasily; there was in the left cavities a little fluid blood: the mitral valve was abnormal; its segments were charged with prominent points, indicating the deposit of atheromatous matter, and between the layers of endocardium the aortic valves were thickened at their bases, and were large and loose. The aorta, free from atheroma, was deeply blood-stained.

The right lung was greatly congested at its inferior part; its upper lobe was dark and very soft, almost pulpy in structure. The structure of the left lung was partly softened, partly solidified; the lower lobe was condensed, and gave out on incision an exudative fluid, unmixed with air; the upper lobe was softened, almost pulpy, and stained with dark blood. There were no pleuritic adhesions.

The liver was generally enlarged, and pale; some bands of softened fibrine connected the organ with the diaphragm by loose adhesions. The tissue was greasy, and so light that it floated in water; microscopically, it proved to have undergone general fatty destruction. The gall-bladder was empty; the duct was free. The peritoneal membrane showed no mark of inflammatory lesion, past or recent. The mucous membrane of the alimentary canal was examined from the mouth to the anus. The gums were of a dead leaden colour and spongy, and the teeth were dark (we attributed these changes to recent

mercurialism); the tongue was white and soft. From the pharynx, the mucous membrane was pale throughout, and showed no mark of vascularity, nor of abrasion. The mucous membrane of the cæcum was darker in shade than the other portions of the tract; but, on inquiry, this was found to depend on submucous pigmentary deposit. The stomach was empty; the remainder of the intestinal mucous surface was lined with a thin mucus; the canal contained scarcely any fæcal matter.

The spleen, which had a broad adhesion to the diaphragm, was soft to absolute pulpiness. The kidneys were large, flabby, pale, speckled, soft, and greasy. The uterus was adherent to the rectum; the os was ulcerated. The bladder was empty and contracted; its mucous surface was tinged red, but there was no sign of abrasion.

After the autopsy there were submitted to me for analysis the following:—

Two bottles supplied by the druggist, one purporting to be a mixture containing quinine, the other a draught containing ipecacuan. I had, further, from the deceased woman's body a portion of the liver, one kidney, a portion of the heart, one ounce of blood, and three drachms of the mucus collected from the alimentary canal.

It is sufficient for me to say, in regard to all these substances, that the most careful analysis did not even supply a suspicion of the presence of any mineral poison, nor of opium, nor of hydrocyanic acid, nor of strychnia. Even mercury, which we assumed from

the state of the mouth had been recently taken, did not show itself. It is but fair to say, however, that I could gain no direct information on the question whether the assumption of a recent mercurial course was founded on fact.

From the evidence gathered from all the above inquiries, my colleagues and myself found an unanimous opinion that the deceased woman had had no poison administered to her; but that with a heart nearly worn out, diseased kidneys, such as we had seen, and suppression of urine for a long time before death, she had sunk under typhoid uræmia. I gave evidence to this effect at the inquest, and the jury returned a verdict in accordance with the evidence.*

A gentleman, sixty-three years old, was driving in

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* I cannot avoid noticing that, in this painful case, all confliction of medical witnesses was removed by the mode in which the *post mortem* inquiry was conducted. The anatomical part was ably carried out by Mr. Miles; the judicial by Mr. Callender and myself, Mr. Marshall aiding with his opinion. We agreed, before any step was taken, to take notes on the state of every organ, and, as each organ was exposed, to determine amongst ourselves its exact condition. Practically, we found this the easiest thing in the world; and, had the case gone on to an assize trial, and had Mr. Callender been called to the defence, he could not on matter of fact have given evidence differing from mine, nor I from that given by him.

I was never more relieved than in feeling that the suspected druggist had (as every suspected man ought to have, by right rather than by courtesy) the benefit of an able observer on his side from the commencement of the inquiry; and I have since made it a rule never to conduct a *post mortem* examination in a doubtful case, without the presence of another professional man, who should be claimed by the opposite side. If this plan were universally adopted, there would be no more unseemly exhibitions of professional disputes in our courts of law.

an open chaise through the village of Mortlake, in 1853; he was observed by his servant, who was by his side, to be constantly drowsy; at last he suddenly seemed to fall into a helpless state, and dropped from the chaise. He was conveyed into a house, and I was summoned. I found him suffering from all the signs of a narcotic poison; the pupils were fixed and slightly dilated. I did not see danger of instant death, and therefore sent for a conveyance that would hold a mattress. I laid the patient down in this, and myself took him to his home, two miles distant, placed him in bed, and watched and treated him until his own medical man, whom I at once sent for, arrived. I suspected this case to be one of uræmic poisoning; and from what I learned from the friends, I became better advised in the same view; but to satisfy myself I drew off some urine from the bladder and tested it: it was largely charged with albumen. Having resigned the care of the patient after a few hours to the medical attendant of the family, I had no personal opportunity of tracing its progress; but I learned that the patient recovered from the attack, which had commenced as above narrated; that three weeks later he suffered again in a similar manner, and died with typhoid coma, the urine being altogether suppressed for many hours before death, and having throughout the whole of the illness been albuminous.

In 1847 I was assisting a country medical friend, for a few months. One morning I went to see

a child that was said to be in "a fit" and violently convulsed. On arrival, I found that the child had been drowsy and unwell for two days; and that, on being called to rise on the third morning, he could not be roused. There was a peculiar dull, almost bronzed appearance of the skin; deep coma, but frequent convulsive movements and cough. The pupils were dilated and fixed. I did what I could, very blind at the moment as to the exact nature of the case; however, I ordered a hot bath, leeches to the temples, and a purge; but it was of no avail: the coma terminated in death after thirty-six hours. A little urine was passed once in that time, and it was intensely albuminous.

While this child was still alive, another child, a sister, was seized; and this time, seeing the child early, I detected a scarlatinal eruption. The redness lapsed into the bronzed tinge in a very few hours, and the same symptoms of uræmia were manifested, with, I regret to say, the same fatal result.

We now gathered, on inquiry, the fact that scarlet fever had been directly imported into the house; two remaining children suffered from it in a less malignant form, and recovered, but not without subsequent albuminuria.

In 1850 I attended a boy, J. S., at Barnes, seven years old, through a severe attack of scarlet fever. The disease was epidemic at the time in the village. About sixteen days after the disease commenced, symptoms of albuminuria presented themselves, and ended in

dropsy; they seemed to have followed an accidental exposure to a cold damp air. Under treatment the child recovered; and when I ceased attendance, a specimen of urine was tested, and found to be not albuminous.

I heard nothing more of this child until January 21st, of the year 1851, when I was summoned in haste, at 10 P. M. I learned that the child, two days before, had been exposed to cold and wet, and that this exposure had been followed by suppression of urine. A little of the urine last passed had been saved for me, and was found to be albuminous. The symptoms were not alarming until the 21st; but when I arrived they were serious enough. The body was cold; the pulse small: the convulsions extreme; the consciousness quite lost. The convulsions were general, involving the muscles of the neck, the face, and the extremities. The pupils were widely dilated and fixed; the breathing was catching and irregular. I at once tied up an arm, and opened a vein freely. The blood at first flowed very feebly, but by-and-bye gave a full stream. It is the fact, that under this depletion the pulse rose and the convulsions ceased. I took not less than sixteen ounces of blood from this little patient without any sign of syncope, and I left him composed and conscious. A purge was afterwards given, and an occasional warm bath. On the 24th the patient sat up, the only noticeable symptom being a slight irregularity of the pulse. On the 1st February there appeared to be no serious mischief remaining; the urine was again passed freely, and was clear of albumen.

On February 24th the child, having, from my last visit on the 1st, been healthy, playing with his fellows merrily, and eating, drinking, and sleeping naturally, was seized, after a hearty meal, with a repetition of his former symptoms, at seven in the evening. I was once more summoned, and regret much that I was induced not again to draw blood from the arm, but to be content with the application of leeches to the temples, and the administration of a scammony purge, with a hot bath. The remedies failed: at one o'clock on the morning of the 25th, the convulsions lapsed into a kind of jactitation, very short and very feeble; and at two o'clock death took the lad.

Twelve hours after the death I made a *post mortem* examination. There was very slight congestion of the cerebral substance, but no engorgement of the sinuses. The lungs were inflated, and quite healthy. The heart was firmly contracted and healthy: the abdominal organs were all healthy, except the kidneys, which were pale and rather large; but I was surprised, on microscopical examination, to discover that they presented no evidence of degeneration nor of disease of vessel or tubule, sufficient to account for the albuminuria and uræmia on the ground of mechanical obstruction commencing in their structure.

I was called in the summer of 1853 to see two children, a boy and a girl, who were taken with convulsions on going to bed. The one was seven, the other eight years old. I found that they had been near to Kew in their play, one of them having told this to

their mother before becoming seriously ill. Both children were now insensible and violently convulsed: their skins were slightly discoloured to a light copper hue all over the surface. The pupils were fixed and dilated. Scarlet fever was present at this time in the neighbourhood; and my mind was perplexed with the doubt whether these children were suffering from uræmia, or from some poison taken by the mouth. After a little time I observed that in one child there was, with the convulsions, a distinct attempt at vomiting. I at once acted on the suggestion supplied, gave a dose of sulphate of zinc, tickled the fauces, and produced in this child free emesis; there were brought up portions of leaves of some plant and small seeds. I turned to the other child, and treated it in the same way with similar results. After the vomiting both children rallied, and next day were well. They recorded plainly enough the cause of their symptoms; they had gathered and eaten the leaves and fruit of the atropa belladonna. I went with them to the place where they had gathered the poison, and saw the plant from which they had eaten.

This case is not irrelevant to the present essay. It indicates the analogy which exists between the symptoms of scarlatinal uræmia and those of poisoning by belladonna, and the possible mistake that may be made in the diagnosis.

I have yet a record of another case where an aged man, suffering from albuminuria, was subjected fre-

quently to attacks of diarrhœa, attacks which were, in truth, compensatory in their natural intentions. During one of these diarrhœal periods, he had a few repeated doses of grey powder combined with Dover's powder, with the effect of intense and rapid salivation. He recovered from this condition slowly. Twelve months later, having been exposed to wet in superintending the repairs of some house properties, he took what he considered to be a cold, and suffered again from copious diarrhœa. Once more the grey powder and Dover's powder were administered, this time on the prescription of a physician, who, having no cognisance of the condition of the patient's urine, treated him simply for diarrhœa. Once more, but with greater rapidity, the mercurial ptyalism was developed, attended with profound coma and frequent convulsive attacks. On the third evening of the illness of this gentleman, I saw him with his own medical adviser, and passed the night in attendance. The symptoms were unmistakable and most severe; the body was generally warm, and at times hot; the coma was profound and the breathing heavy; the pupils were slightly dilated, and fixed. The effects of the mercury were such that the gums were enlarged throughout the whole of their surface, the lips were swollen, the teeth were loose, and saliva was constantly exuding from the mouth. About every two hours, the scene of deep repose was changed by the occurrence of a violent convulsive paroxysm. The convulsion commenced in the muscles of the face, then extended to those of the neck

and trunk, and at last to the limbs. I should compare the attack rather to a shaking of the whole body than to an epileptiform seizure; for in epilepsy some one part of the body is usually fixed, while the limbs are cast about; but here body and limbs together were ruthlessly disturbed as by a general and evenly disposed shock, in which not a single muscle escaped. The attacks lasted about fifteen minutes and then subsided; the coma, which had never relaxed, remaining again sole lord of the evil. The pulse was full and slow—even during the paroxysm this did not vary: the respiration was slow and deep, but the vesicular murmur was clear. The bowels were at this time constipated, the mercurial opiate having done its astringent mission only too well. At times a little urine was passed in a semi-voluntary manner, and on examination was found to be so charged with albumen, that it formed a coagulum shaped to the iron spoon in which it was heated. When a firm grasp was made over the loins, the patient shrank as though this part were tender to the touch, but elsewhere no expression of pain was elicited by pressure. The symptoms being urgent and apparently hopeless, I advised with my medical colleague the free abstraction of blood. This agreed to, we cupped freely over the loins; and, the coma remaining, we next removed twelve ounces of blood from a vein in the arm. It is the fact that, while this blood was flowing, the patient for the first time in seventy hours became sensible, sat propped up by pillows, recognised all his friends, remained conscious after the venesection

for several hours, took some beef-tea, and arranged certain business matters which had been pending for his decision. Early on the fourth morning, the friends of the patient called in a third opinion, that of an eminent physician since deceased. It was unhappy that, when he came, while he agreed with me as to the pathology, he and my other colleague differed from me in respect to treatment. I insisted on following up the venesection by a gentle series of saline purgatives, containing small doses of colchicum; being guided in this suggestion by the belief that the original diarrhœal flux was an eliminative act, and ought to be reinduced. My friends thought otherwise; and, as the difference between us was very wide, and I was in a minority and the junior of the consultants, I thought it best, as it was my duty, to retire in an amicable way from a case, in the management of which I could not conscientiously take part on a common understanding. On the evening of the day in question, the patient relapsed into the comatose condition, and on the following morning died in one of the convulsive paroxysms. It is possible that the same result would have occurred under any circumstances, for an active purge might, as my colleagues thought, have induced a fatal diarrhœa; but, taking all in all, I have always regretted that that risk was not accepted, and that an effort was not made to relieve steadily the congested imperfectly acting kidneys by exciting a demand on another emunctory surface.

I have thus adduced eight cases; in six of which

there was positive uræmia; in two, simulated uræmia from the effects of a narcotic poison. The uræmic patients were of widely different characters, presenting gradations from infancy to old age. In the three youngest examples, the disorder was a result of scarlatina; but in one of these the original disease had long passed away, while in the remaining two it had scarcely laid its hand on its victims. In the woman of middle life, the uræmic state was the direct result of alcoholic intemperance, syphilis, and perhaps mercury—trinity of disease-makers. Of the two men advanced in life, both suffered, previously to the final attack, with manifestations of intestinal derangement; and one was twice salivated by doses of mercury, which even to children are considered as innocuous. All the six cases ended fatally, the fatal symptoms being closely identical. In each example the comatose symptoms, from their first appearance to their termination in death, were included in a period of time, the extreme duration of which was eighty-three hours. In four of the cases the fatal illness was with the first attack, and in two with the second. In these last two cases, recovery from previous seizures had occurred; in one apparently under the direct influence of treatment, in the other spontaneously, and in spite of mercurial ptyalism. The cases thus given, selected from many others, afford illustrations of all the typical forms of uræmia.

DIAGNOSIS OF URÆMIA, ABSOLUTE AND DIFFERENTIAL.

The symptoms of uræmia are so closely allied to those presented in other forms of disease, that it would lead me into an almost impossible task to attempt a differential diagnosis applicable to every case. I shall therefore endeavour first to establish, as near as can be, an absolute diagnosis. Such differences as may exist between uræmia and certain other allied diseases may then be noticed with rapidity, and with a certain degree of precision.

Absolute Diagnosis. I should include in the following symptoms the nearest verbal description of simple uræmia.

There are almost invariably some premonitory signs. These consist of sleepfulness—an irresistible desire to sleep at almost any time and place. With this there may be paralysis of sensation in extreme parts, as in the hands, lips, or feet. In one case with which I was acquainted, this sign frequently occurred long before the decided and final attack: the patient said he felt as if he were treading on wool, and his feet seemed to give way, although he could stand on them steadily and use them as well as ever, when he made an effort to that effect. Or, again, there is a confusion of the mind, transitory in its character, and conveying the impression that objects near at hand on which the sight is fixed are receding or dissolving. There is no vertigo, and no oppression with this peculiarity, but a feebleness and listlessness. There may also be vomiting, sometimes very severe; and in one

example I knew the ejected matter to be charged with blood, the hæmatemesis occurring at least twice as a premonitory sign. There may be occasional relaxation of the bowels, which relaxation gives relief and renders the mind clearer for a time, if it be not too profuse and too continuous. Lastly, there is very commonly a peculiar fœtid condition of the breath, a sickening odour more like that caused by sulphide of ammonium than aught else. The breath in this condition yields ammonia largely.

Persons affected with a tendency to uræmia suffer exceedingly from cold; they feel the influence of a fall in the thermometer acutely, and the symptoms of the uræmic coma have often their date from such a declination of temperature. The action of cold in this case is evidently upon the skin and pulmonary membrane primarily, and by reflection on the kidneys, leading to congestion and obstruction. Again, these sufferers, more than others, feel the effects of a hearty meal, especially if that consist largely of nitrogenous foods. I believe that in many cases the so-called apoplexy occurring after a meal, and remaining for many hours before ending either in death or recovery, is uræmic in character.

Patients disposed to uræmia are further dangerously susceptible to the action of certain medicines. The smallest mercurial dose seems sufficient in them to shut off the renal secretion and excite the acute uræmic paroxysm. Mercury, however, is not the only drug productive of this evil. I have seen, with exception of the ptyalism, exactly the same effects

follow the administration of an ordinary dose of opium. I cannot conceal my belief, indeed, that in many cases where opium has seemed to destroy life when given in very small doses, the "idiosyncrasy" assumed to have been present has consisted in a condition of kidney, during the existence of which uræmia in its acute form might have stood prognosticated, had the earlier symptoms been known.

The acute symptoms of uræmia come on either with coma or convulsion, or both combined. They are usually sudden in their intensity, and are preceded by suppression of urine, or by constipation, or by a chill by which the eliminating function of the skin is suppressed. There is often active convulsion; but I have seen death occur from uræmia without much convulsive movement. Nay, I have seen an animal die from artificial uræmia without active convulsion, though this is an exception. There occasionally will be squinting in the course of the acute paroxysm, but this is not the rule. The state of the pupil varies; but it usually is largely dilated, and is insensible to light, and fixed; the countenance is dusky; the skin is unimpressionable and cool, with short exacerbations of heat.

If the coma be profound, the pulse is slow and intermittent, or irregular in periods. When I say that the pulse is irregular in periods, I mean that, counted over two spaces of time of equal duration, it shows a difference in those two periods. We find the pulse during one minute at 60; we wait a minute and take it again, and although the relation of beat to beat

seems the same, we find that in the course of the second observation five, ten, or even fifteen pulsations have been gained or lost. This form of irregularity of pulse is not peculiar to uræmia, but is common in it, and is inevitably a bad sign. Traced to its cause, it is found to depend on a corresponding irregularity of the respiration, for in nearly all forms of slow narcotic poisoning the heart is obedient to the respiration. The respiration is, from the beginning to the end, irregular and imperfect; there is not dyspnœa, but uncertainty; no obstruction to air, but a destroyed balance in the respiratory movements.

Much has been argued as to the condition of the expired air in uræmia; Frerichs has stated that the breath is largely ammoniacal in this state. Other observers have denied the position. In fact, both are right and both are wrong. The true position is simple. In all persons there is an exhalation of ammonia by the breath, varying in amount. In uræmic persons, in their ordinary health, there is, I believe, always an excess of ammonia in the breath; for in them the lungs are supplementing the kidneys. If, then, in any case there be sudden suppression of the excretory power of the kidneys, there may be an excess of ammonia in the breath. I have unmistakably seen examples where this excess was clearly manifested—so clearly that the exhaled vapour gave distinct fumes to the hydrochloric acid rod, and even changed the colour of reddened litmus. On the other hand, there are cases where the very cause of the

development of the acute symptoms lies in the fact that the compensating eliminative function of the lungs has become suppressed: in such state there will be no ammonia found in the breath; it would be a saving clause if the alkali were there.

I have but one word more to say respecting the absolute diagnosis. It relates to the degree of consciousness manifested by uræmic patients in the extreme state.

There are periods when the coma is so profound that nothing arouses the sufferer. There are, again, other times when he arouses of himself, or is quite capable of being aroused, and of speaking or attempting to speak, and of sitting up, and of swallowing foods or medicine, and of acting altogether with a considerable amount of intelligence.

The symptoms above described, taken in all their entirety, are sufficient to give a correct picture of uræmic disease. But it is worthy of remark that there are special symptoms in different cases which, without removing the idea of the general fact that the arrest of the function of the kidneys is the cause of the symptoms altogether, suggest forcibly either that the poison is different in certain instances, or that, being the same in all, it acts variously in different persons, by difference of dose, or by peculiarity of effect on the individual organism. Thus in one case the pupil may be dilated, in another natural in size; in one patient there shall be active convulsions, in another no convulsion whatever; in one there shall be marked derangement of the bowels, in another no intestinal irritation shall be presented; in one there

shall be vomiting as an early symptom, in the next no indication of such an effect. These distinctions, all of import, are nevertheless secondary; since, in every case, they are included in two common symptoms—excretion of albuminous urine, and coma.

Differential Diagnosis. Uræmia simulates in some particulars so many of the diseases in which coma is the leading symptom, that an attempt to enter into the distinctions that mark it off from every analogous disorder would be interminable. In some instances, too, the task would be an exceedingly difficult one, unless considerations relating to the previous existence of albuminuria were admitted into the argument. Thus, for example, the diagnosis between uræmic poisoning and belladonna poisoning is so difficult, that, in a case where the history of the patient was unknown until the occurrence of coma, I for one, after having witnessed both classes of toxæmic disease, would scarcely be able to note a trustworthy sign on which to build a decided opinion. It is obvious, in fact, that the poisons producing the two classes of symptoms are well nigh identical in regard to their effects. Here, therefore, we must rest on the course which the symptoms take, and on such occurrences as the vomiting of suspicious matters, for a guide to diagnosis, rather than on any particular or exclusive symptom.

From epilepsy, uræmia is distinguished by the tendency to diarrhœa, and, above all, by the suppression of urine. Distinctions may also be drawn at the time of the acute paroxysm; for the convulsion of

uræmia is not of the same tonic character as in epilepsy; the asphyxia is not so marked, and the coma is more prolonged.

At one time in the history of medicine, uræmia constituted one of the varieties of coma called generically "apoplexy." The unlearned as yet call uræmia apoplexy. From true cerebral apoplexy, however, uræmia is distinguishable by the facts that the skin is less hot, the stertor less marked, the blowing expiration less frequent, the convulsion more defined, determinate, and paroxysmal, and the suppression of urine more certain.

From poisoning by opium, uræmia is to be distinguished by the following particulars. In uræmia, the pupils are generally dilated; the patient may become quite sensible during the acute attack, and then profoundly relapse; the bowels are often free; the urine is scanty or absent; the breathing is free from stertor; and the convulsions are active. In opium poisoning the pupil is contracted; the patient remains unconscious, or, recovering his consciousness, remains conscious; the breathing is stertorous; the urine free; and the muscles paralytic rather than convulsed.

In fine, in establishing, in a doubtful case, a differential diagnosis between uræmic intoxication and its analogues, we must depend most on the previous history of the patient, and on a correct recollection of the absolute symptoms by which uræmia is manifested. The careful practitioner will then rarely be misled. If, however, a preceding history cannot

be gleaned, if the symptoms be obscure, and if urine be not passed by the patient, it is an excellent point of practice to draw off by a catheter the urine that may be contained in the bladder, and test for albumen.

The presence of ammonia in the breath, looked on by many as a definitive indication of uræmia, is not, according to my mind, of any peculiar diagnostic value; inasmuch as ammonia is presented in the breath in the natural condition, and is expired in excess in many other diseased states than uræmia: thus, in the coma of typhus and of typhoid fevers, an excessive ammoniacal exhalation from the breath is a common symptom. Further, there are examples of acute uræmia where the ammoniacal exhalation, common to the patient antecedently to the attack, is suspended during the comatose state. Without going so far, therefore, as to say that the presence of ammonia in excess in the breath is not to be taken with other indications as a corroborative sign of uræmia, I should not let it rank as a positive symptom for differential purposes.

COMPLICATIONS OF URÆMIA.

I have tried, so far, to give the diagnostic history of uræmia in its simple form; but it happens often that there are certain complications which present symptoms delusive in character, and calculated greatly to deceive him who has not his eye on the unities of diseased action. These complications consist generally of inflammatory or pseudo-inflammatory outbreaks, ac-

companied by pain if the patient be at all anxious, perhaps by swelling, certainly by effusion. These inflammatory outbursts may occur anywhere, as if in fact by accident; in the peritoneum, in the pleura, in the tissue of the lungs, in the bronchial mucous tract, in the alimentary mucous tract, in the encephalon. They may vary in character; they may be acute in acute uræmia, or in chronic uræmia subacute and very gradual in progression. They may prove eventually the immediate cause of death. The day will come when all these forms of inflammatory lesion will be classified under one head, as derived from the primary uræmic diathesis. But to mention these inflammatory complications, to notice their symptomatic connection with the uræmic state, is sufficient for this moment. I shall treat in a subsequent section on their production.

PATHOLOGY OF URÆMIA.

In all cases of uræmic intoxication, whether preceded by chronic renal disease, or occurring suddenly during an acute inflammatory attack, or induced in an animal by removal of the kidney, the blood assumes various physical and chemical peculiarities, essentially characteristic of alkaline or alkaloidal toxæmia. In the first stages of the uræmic attack, the blood truly may assume the inflammatory cast; *i. e.* it may be buffed and cupped. I saw this markedly in one instance where uræmic coma came on in a harvest-labourer, who, after a night's exposure to a damp cold air, was seized with inflammatory dropsy, followed

quickly by uræmic coma; and I saw the same in a case of scarlet fever. But this inflammatory condition soon subsides, even if it occur; and the blood then undergoes what might very correctly be termed alkaline decomposition. The tendency of this blood is to remain fluid after it is removed from the body, or, if it separate at all, to separate into loose shreddy masses of fibrine. If to the fluid blood milk of lime or baryta be added, there is rapid coagulation with evolution of ammonia. The corpuscles of uræmic blood undergo also great modifications. They are crenate at the edge, they take an oval form, show little tendency to coalesce, and are reduced in number. This diminution of red corpuscles, first observed by Christison, I have been able to confirm in two cases on the human subject, and in an experimental case on an inferior animal. I have noticed the same diminution of corpuscles after poisoning by belladonna.

There is also an excess of serum in proportion to the solid constituents in uræmic blood; and often a low specific of the serum, owing to a deficiency of albumen. The fibrine in the later stages seems to become decreased; but whether this decrease is absolute or relative I am unable to say. Certain it is that uræmic blood, in which the fibrine is apparently so far absent that spontaneous coagulation is impossible, will coagulate readily enough on the addition of milk of lime, baryta, or a weak solution of potassa cautiously added. It is possible, therefore, that the fibrine is not actually deficient, but is held

in solution by the presence of an alkaline body, which is liberated as an ammonia on the addition of a displacing agent.

In a case recorded by F. Simon, the serum exhibited a remarkable milk-white turbidity. This colour, which was not caused by fat in a state of suspension, was found to depend on the presence of numerous minute solid granules, which on collection and inquiry were found to be insoluble in alcohol and ether, but soluble in dilute acetic acid, from which they were separable by ferrocyanide of potassium. Hence Simon concluded that they were particles of fibrine. The same experimentalist found hæmatoglobulin more abundant in the hæmatin in cases of uræmia, than in ordinary cases. It varied from 8 to 9.5 per cent.

The amount of urea in the blood is increased. This fact, asserted by Christison, Simon, and all the earlier writers, but disputed by other succeeding authors, is now satisfactorily proved. The experiments of Hammond on the increase of urea in dogs, after extirpation of the kidneys, remove all doubt. In one of his carefully conducted inquiries, 100 grammes of blood removed from the jugular vein before extirpation of the kidneys yielded 0.026 gramme of urea: whereas, twenty-four hours after extirpation, 100 grammes of blood from the same vein yielded 0.083 gramme of urea.

From these peculiarities of blood, we may pass to the pathology of other structures. Effusions are common during uræmia: they must be so, for, in condi-

tions where the functions of the kidney are suppressed, and albumen is an excrete, not only is there accumulation of water in the tissues, but there is decreased specific gravity of blood, which, in proportion as it exists, induces exudation by incapacitating that fluid from eliminating its water. This result of uræmia—exudation—occurs in divers parts of the organism; in the cellular tissue, in the serous cavities, in the bronchial mucous tract, in the ventricles of the brain. With the mere serum thrown out, pseudo-plastic material is sometimes admixed; and, after death from uræmic coma, these exudative products may constitute a marked feature in the morbid representations. They are to be looked upon as sequences; and, although they may materially have influenced the course of a case, their secondary character should invariably be recognised by the philosophical student.

Whether effusion exist or not in uræmia, there is always more or less of congestion of the vascular organs. In the earlier stages this frequently leads to diseased states of special organs, inflammatory in appearance. In later stages the congestion becomes universal; for, as we shall see by and by, in comparing the mode of death in uræmia with asphyxia, that while the congestion commencing at the kidney is for a time partial only, extending to the lungs it is reflected through the whole of the soft tissues. Hence, after death from uræmic coma, we almost invariably find congestive enlargement of the liver and spleen, intense congestion of the lungs, with distension of the sinuses of the cerebral membranes, and congestion of

the minute vessels of the brain itself. For this same reason the muscles are often dark and filled with blood, and the inner surface of the arterial system is deeply stained.

It is worthy, too, of special remembrance, that the congestive condition above described may extend to the alimentary mucous surface in any part of its tract.

This fact, of which I have given one illustration (*vide* pp. 134-5), is important, inasmuch as the congestion of the alimentary tube is usually followed by exudation of serous fluid containing urea. Mixing now with the secretions natural to the canal, the urea is transformed partially or altogether into carbonate of ammonia, which acting in turn as an irritant, produces, when present in excess, distinct gastro-enterite with diarrhœa and vomiting as symptoms, and an inflamed mucous membrane as the visible morbid upshot. Thus in the train of acute uræmia we may have all the results of an irritant poison. We may have gastro-intestinal inflammation, or peritoneal effusion, or both combined. We may have congestion of lung, pleural exudation; cerebral congestion, ventricular effusion; deep staining of the endocardial membrane, and equally dark staining of muscular fibre. We may, I say, have all these obvious lesions, or we may have none of them; for if, on the one hand, the suppression of the kidney be immediate and perfect, death may take place purely from a rapid disorganisation of the blood, owing to accumulation of water with the urea; or if the arrest of the renal

secretion be slow, so that time is given for the exudation of serum containing urea, the decomposition of the urea is the result, and of that decomposition all the lesions which have been pointed out are necessary sequelæ.

It would lead me into a subject foreign somewhat to this essay, to discuss at any length the morbid conditions of the kidney with which uræmia is related. Some authors have endeavoured to connect uræmia with special lesions of the kidney. My own experience is to the effect, that uræmia may succeed on any acute or chronic obstruction in the renal organs. After scarlet fever, in addition to the congestion, the condition met with, perhaps in all cases, is exudation into the tubules and epithelial desquamation. In other cases, atrophy is the morbid representation; in a third and more numerous class, enlargement with granular, fatty, or waxy degeneration. In a fourth modification (of which the case of the boy J. S., whose history is given at pp. 142-4, is a type), the kidney may present no sufficient lesion to account for the symptoms and their consequences. In these examples it is probable that there is some latent and undiscovered lesion of the nervous centres or of the renal nerves, by which lesion the secreting power of the kidney is reduced or estranged; for we know that all the effects of uræmia are producible by simple division of the renal nerves.

The pathology of the urine varies with the state of the uræmic patient. There may be periods when persons who are the subject of renal obstructive dis-

ease have their urine free of abnormal constituents, but during uræmic coma, on which only this essay treats, there is invariably a large presence of albumen. In this respect, both in acute and chronic cases the rule is the same; but in other respects there may be distinctions, not only between one case and another, but in the same case at different periods. For example, in acute cases, such as those which occur early in scarlatina, the urine is albuminous, although to external appearance it is pyrexial, *i. e.* high coloured, small in quantity, and charged with red sediment; from ignoring this circumstance of pyrexial albuminous urine, scarlatinal uræmia is often overlooked until it is represented by deep and unmistakable coma.

With the presence of albumen in the urine in these stages, there may be a decreased amount of urea; but this is not an absolute rule, for occasionally the urea is temporarily increased. There is a deficiency of chlorides; and, sometimes in addition to the foreign element albumen, there are blood itself, fibrine, casts of tubes, and mucus. As the disease advances, the albumen increases in quantity and the urea is decreased, but without any absolute relationship in respect to the proportion of either substance.

In cases where there has been long standing kidney-disease, with uræmic coma supervening, the quantity of urine is usually scanty; the appearance of the fluid closely resembles thin beef-tea, or thin white of egg emulsion; the reaction is neutral or very feebly acid; the amount of urea and uric acid

is decreased; the chlorides are decreased; and as unnatural constituents there are albumen, casts of tubes, epithelial cells, and mucus. These conditions of the renal secretion are those common during the absence of pyrexial disturbance dependent on local inflammatory lesions. In pyrexial states the urine may become highly coloured, and may even give an abundant proportion of urea.*

CAUSE AND CAUSATION OF URÆMIC COMA.

We know as a primary fact that uræmic coma is the result of arrested secretion of urine.

The proposition is proved by the direct experiment performed on inferior animals, of arresting the urinary secretion, either by removal of the kidneys, by ligature of the renal vessels, or by division of the renal nerves. When any one of these experiments is carefully conducted, the symptoms and pathology of uræmia are produced with as much accuracy of detail as can be desired.

The symptoms caused by suppression of the function of the kidney (I speak from my own experimental observation conducted on dogs) vary somewhat according to the degree of the operation. If both kidneys be removed from action, uræmia is an inevitable fact. If one kidney only be suppressed in function, uræmia in the majority of cases is again the fact; but occasionally there is this exception, that the

* For the most perfect history extant on the condition of the urine in acute and chronic uræmia, I refer the reader to Dr. Parkes's work *On the Urine*, chap. v, pp. 376-394.

animal recovers without the occurrence of any serious mischief. Granting the accession of uræmic symptoms after either operation, they are not usually presented for ten or twelve hours. The first symptom then observed is a rigor, which subsiding leaves the subject sleepy and motionless. For some time, however, he may be roused without much difficulty; but on the occasion of all such attempts he is, as a general rule, disposed to vomit. He will refuse food, but will often partake freely of water. Dogs in this state never express anger nor sign of acute pain. At intervals there are paroxysms of convulsion, but very subdued in kind, and apparently occurring during periods of oblivion. After the development of the first rigors, the temperature of the body rises; I have seen the thermometer in the mouth rise, for example, six degrees beyond that at which it stood before the operation. The respiration, so soon as the somnolency is established, becomes markedly irregular; it will vary ten or even twenty in number per minute; at the same time, it rarely exceeds fifty inspirations and expirations per minute. Corresponding with this irregularity of respiration, the action of the heart is equally unbalanced. There may be a variation of so many as twenty and from that to thirty beats per minute; and here too, as in the human subject, this peculiarity may be marked, that the irregularity shall extend over a long space, and so observed shall become very obvious, while per beat it shall not stand out at all distinctly. If these symptoms have been induced by the removal of

but one kidney, the probabilities are, that once or twice in the first eighteen hours after the operation a small quantity of urine will be passed. On examination the fluid will be found albuminous, and perhaps containing blood or fibrine.

The symptoms thus detailed usually continue for twenty-four or thirty hours without any striking modification. At last the gap in the respirations becomes more distinct, an interval of even ten seconds intervening between two inspirations, followed by a series of rapid expirations. At this stage the pulse will cease to beat altogether during the intermission of the respiration; but as the hurried breathing is remanifested, the action of the heart will follow, and be so quick as scarcely to admit of being counted. The temperature of the body, previously raised, now begins to fall; convulsive actions give way to an entire passivity and prostration of muscle. The coma continues, and at last death takes place as from universal muscular paralysis. The heart seems to me to die first.

After death, *post mortem* rigidity is fully developed. The blood in both systems is usually dark, and is often either fluid or in a soft jelly-like condition; it coagulates slowly if found fluid, or may not coagulate at all. There is great vascular congestion of the lungs, the abdominal viscera, and the brain; and sometimes there are effusions of serum into the serous cavities, in which effusions urea may be found. The muscles are dark; and the endocardial surface may be stained deeply. The alimentary tract may also show signs of redness and congestion.

Thus between uræmia as it occurs from disease, and as it is induced by the synthetic process, there is identity both as regards symptoms and pathology. The proposition founded on this identity accepted, we have next to ask—What is there in normal urine, (for we may take the urine as normal) capable of producing the toxæmic symptoms of uræmia?

Setting aside hypotheses which have been invented merely as verbal trophies, there are certain speculations which demand our careful attention. Let us note them.

The earliest accepted view is that which attributes the symptoms of uræmia to retained urea, which in fact accepts urea, or, in more correct words, cyanate of ammonia, as a narcotic poison. This view has been by some eminent authorities set aside, to be recently revived and most ably advocated by a distinguished American physiologist, Dr. Hammond.

Another hypothesis is one put forward by the justly eminent Frerichs. He argues that the retained urea, undergoing decomposition in the body, is converted into carbonate of ammonia. The carbonate of ammonia thus produced is, according to Frerichs, the toxic agent.

There have been at various times certain other hypotheses put forward, which have more or less ignored urea as a producing cause, either in a direct or indirect sense, and in substitution for it have suggested as the poisonous agent other constituents of the urine; as the water in excess in the blood, or the constituents of the urine altogether. Into all these later

hypotheses a certain vagueness enters, which throws us back at once on the first and fundamental proposition, and leaves us with that alone in our possession as a fact.

Men who know nothing of the immense difficulties of conducting physiological researches may, I am aware, be ready with their criticisms; and may twit their more inquisitive and laborious compeers on their failures in satisfactorily settling a question on the surface of it so simple. But, in truth, in actual practice the question is one of immense difficulty, requiring for its solution not only actual experiment, but experiment so devised as to give, on the one hand, direct affirmative or negative replies, and on the other hand to be so far free of side influences, that the facts which are either affirmatively or negatively given shall not admit afterwards of interpretation by any new reading of their cause.

Let us, in considering this question, divest our minds for the moment of all thoughts respecting the immense interests which attach to its solution; let us throw aside every prejudice or weight of past learning that may so easily beset us; let us make no ambitious strain towards divining the riddle; let us use no oracular obscurities, chemical or physical; let us ask the aid of bare common sense, rest on it alone, and, at the risk of being considered puerile, question nature with the simplicity of a child.

We know now with tolerable accuracy the composition of the urine; and we can prove therefore that a certain fluid, the constituents of which we can

analytically read off, is capable of producing the specific symptoms of uræmia. We turn naturally from this knowledge to an examination of the constituent parts of the urine, and ask, respecting each of them, one after the other, can this be the poison? We discard several at once, from a comprehension of the fact that the symptoms they produce when present in excess in the body are not of a character analogous to the symptoms of uræmia: by this principle we exclude, with safety, uric, hippuric, and phosphoric acids; the chloride of sodium; the sulphates of potassa and soda; the lime and magnesian phosphates; and the iron. We have, again, the cells of epithelium and mucus derived from the bladder and mucous tract, which to a certain amount are present in every specimen of urine. We may exclude these organic products as the cause of uræmia; for, although it has been stated that urine containing them is poisonous on injection into the veins of healthy animals, while urine from which they have been freed by filtration is not poisonous on such injection, it has never been shown that the symptoms produced by the injection of unfiltered urine are those of uræmia. Further, it is obvious that in uræmia arising from obstruction in the renal vessels, or from any other cause by which the secretion of urine is prevented at the *source* of the secretion, the epithelial cells and mucus derived from the conveying mucous channels beneath the source, cannot for a moment be considered as the cause of the symptoms.

Thus by the exclusion process we are driven at last to these constituent parts of the urine, the *water*,

the *urea*, the *creatine* and the *creatinine*. Are they by retention the cause of uræmic coma?

There can be no doubt that water in excess in the blood produces symptoms similar to those presented in some forms of uræmia. Some years ago I demonstrated this fact by producing in animals artificial dropsy. The facts elicited by these inquiries will be found in my essay on the *Coagulation of the Blood;* but the summary of them may be briefly stated as strictly in place here.

When an amount of fluid, equal to a fifth part of the weight of an animal (dogs were used exclusively in my experiments) is injected into the peritoneal cavity, death is the result. Very shortly after such injection the animal becomes drowsy, and eventually quite comatose; the coma advancing in proportion as the fluid is absorbed into the blood. The animal may now and then, at long intervals, get up and attempt to walk with a drowsy tottering gait, but quickly reclines again and sleeps on. The ordinary period of death is from ten to twelve hours after the operation; the death is so quiet that it seems like a continuation of sleep.

If the amount of water injected be less in weight than a fifth of the body of the animal, the symptoms induced are identical in character with those already described, but the animal gradually recovers from his somnolency; he remains feeble for a few days, refuses food and drink, passes large quantities of urine, presents loud anæmic bruit in the neck, staggers in his walk, and is very cold over the whole of the sur-

face of the body. The first indication of convalescence is a desire for food. At least three days elapse before the animal begins to run about healthily; but when recovery is set up it proceeds rapidly, and no ill effects seem to follow.

Such are the general symptoms of an excess of water in the blood; but there are other special modifications in the blood itself possessing great interest. If blood be drawn while the animal is suffering from the effects of the injection, that fluid is found thin, of red colour, and very feebly coagulable. In extreme cases the property of coagulation is lost altogether; and if it be attempted to extract fibrine by agitation of the blood, the experiment fails. Even the coagulation of the albumen by heat is feebly presented. The blood consequently flows with great ease from a wound; and the anæmic hæmorrhagic diathesis is set up. The red corpuscles are much changed; they are greatly enlarged, and assume an oval form. As recovery occurs, these abnormal conditions gradually pass away; but for many weeks the corpuscles retain their diseased outline and character. In a case of extreme anæmia in the human subject, my friend and colleague, Dr. Leared, observed the same modifications in the blood-corpuscles that I have described as occurring in the dog after the production of artificial anæmia and dropsy.

If the symptoms thus induced by the introduction of water into a healthy organism, be compared with those observed in acute uræmia, a certain analogy at once strikes the mind; *i. e.*, there are the same de-

cided prostration and coma. But here the analogy ceases. There is no period during which the heat of the body is increased; there are no convulsions; there is no dilatation of the pupil; there is no vomiting; there is no stertor in the breathing; and lastly, there is none of that irregularity of the respiration and circulation which so peculiarly distinguishes uræmic intoxication.

On the other hand, there is one symptom which is exhibited after the injection of water into the organism which is not present in uræmia, or at all events is not necessarily presented; I mean anæmic bruit.

Taking the symptoms, then, as derived from both sources—from true uræmia on the one side, and from surcharge of water on the other—we are bound to altogether exclude from the argument the hypothesis of excess of water as the cause of acute uræmia. When we admit that the coma of uræmia is possibly in some cases increased by suppression of mere aqueous secretion, we admit honestly and necessarily an intensifying source of one particular symptom; but we fail to trace anything more than a coincidental fact, even in this concession.

From the consideration of water as a cause of uræmic coma, we pass to urea. Is this a poison, and is it *the* poison?

The answer to these questions is to be found only by experiment on inferior animals. To solve the question by this means many hands have been employed, and different opinions have been arrived at. An absence of uniformity in the mode of conducting

the inquiries has, I think, led to the differences observed.

Administered by the mouth to an animal, urea can scarcely be called a poison, unless the dose be enormously large. A dog weighing ten pounds takes a drachm of urea with little result; but in rabbits the dose named excites vomiting and convulsion, and sometimes leads to death. Taking it all in all, the method of experimenting by administering the substance by the mouth is most imperfect. In some cases the compound is decomposed in the stomach; in other cases it is in part absorbed into the blood, and passes off by the kidneys; in many instances it is vomited in great portion soon after it is swallowed, or it may pass off by the bowels in a brisk purge; however it is disposed of, it does not definitely and clearly bring out the true uræmic symptoms as they are elicited on suppressing the renal function by extirpation of the kidney.

Administered by direct injection into the veins, urea can be made to induce symptoms essentially uræmic. Dr. Hammond records an experiment in which the injection of five grammes of urea, or 77.17 grains in solution in water, into the jugular vein of a dog weighing 38½ pounds, at 10 a.m. produced after the lapse of 45 minutes slight spasms of the muscles of the eyelids, and in 50 minutes severe general convulsion; the convulsions continued for fifteen minutes with great violence, when coma followed and lasted until 6 p.m., when the animal died. There was no excretion of urine after the injection of urea. The breath

was examined every hour for ammonia, but at no time was it detected. It was, however, found in the blood last drawn. There was neither purging nor vomiting. Immediately after death the following *post mortem* results were yielded:—

"The substance of the brain appeared to be perfectly healthy; but there was considerable injection of the vessels of the meninges. The ventricles contained about 15 cubic centimetres of serous fluid. Urea was detected in this by chemical analysis, and by microscopical examination. It was likewise found in the blood from the sinuses.

"The vertebral canal was laid open, and the spinal cord examined. Its substance presented a normal appearance, but there was some congestion of the vessels of its membranes.

"The chest contained a small quantity of serous fluid. The lungs were congested, but were otherwise healthy. The heart was of normal size, and did not appear to be in the least diseased. It contained a considerable quantity of fluid blood; 100 grammes were collected from it and the large vessels. The urea in this quantity amounted to 0.873 of a gramme.

"Upon microscopical examination of this blood, the red corpuscles were found to present a crenated margin, and to be in decidedly less than the normal quantity. The white corpuscles were very much increased in quantity; as much as in well-marked leucocythemia.

"The cavity of the peritoneum contained a small quantity of serous liquid. The membrane was in places slightly congested.

"The liver was healthy in appearance; but the spleen was considerably enlarged, and contained much more than the normal quantity of blood. The tissue of this latter organ, when examined microscopically, was found to present several important deviations from the normal structure. The Malpighian corpuscles were almost entirely absent, and there was a very great increase in the number of parenchyma cells. These latter were much larger than I (Hammond) have ever found them in the spleen of the dog. The red blood-corpuscles in the splenic blood were generally aggregated in groups, and were of irregular forms.

"The stomach was opened, and presented nothing abnormal. The contents, consisting of mucus with a few pieces of bone, were of alkaline reaction, and contained both urea and ammonia, the latter in considerable amount.

"The kidneys were enlarged and very much congested. Upon cutting into them, the blood poured out from innumerable orifices. There was no obstruction to either the renal arteries or veins that was discovered after death. The tissue of the kidneys, when submitted to microscopical examination, showed excessive congestion of the capillaries, and enlargement of the Malpighian bodies. Into many of these latter extravasation of blood had taken place, and the tubes were gorged with this fluid.

"The bladder contained a small quantity of bloody urine."

There are not in every case such decided results, on the injection of urea directly into the circulation,

as are above given from the experiment by Dr. Hammond. Indeed, the effects which resulted from his experiment may be considered due to the congestion of kidney produced, as much as to the direct influence of the urea injected; for the fact is that urea, after being injected into the veins, is carried out of the body by the kidneys with such rapidity that its toxæmic properties are not elicited, unless some obstruction be induced in the secerning organ. Impressed with a knowledge of these facts, I have recently instituted a different series of experiments, by injecting the urea in watery solution, not into the blood, but simply under the skin or into the peritoneal cavities of animals. The following is an epitome of the results obtained.

Injected in solution into the dorsal cuticular sac of frogs weighing from 600 to 650 grains, urea is a poison, fatal or not according to the dose. Thirty grains of urea dissolved in a drachm of water produce profound coma and prostration in twenty minutes, with death within the hour. There is great collapse of the tissues, but there are no convulsions, death being too rapid for this effect. Previously to death there is no indication of ammoniacal evolution from the animal, but afterwards the putrefactive changes are very rapid.

When doses of fifteen grains are injected in like manner into similar animals, the symptoms do not appear within a period of half an hour after the injection, and life is prolonged nearly to the second hour. In other respects the symptoms are the same.

From doses of five to ten grains, injected into frogs of the weight already mentioned, severe symptoms are elicited, but with recovery in the end. Symptoms of prostration and coma appear in from two to three hours, and remain little abated for four hours or four hours and a half; there is usually convulsive movement in these cases towards the end of the third hour, such movement seeming to augur recovery. The bodies of the animals thus treated assume externally a dark aspect; and, while recovery is taking place, the whole skin is covered with a frothy excretion.

In young warm blooded animals, as young rabbits, the injection of sixty grains of urea dissolved in a hundred and twenty grains of water is followed within an hour by tremors and coma; the tremors soon lapse into active convulsions, with rolling on the side, and constant twitching of the ears. In three hours the coma is most profound, and the convulsions more feeble. Death occurs about the fourth hour after the injection; while recovery, if that take place, commences about the same period. The pupils are fixed and dilated, and the breathing is very irregular.

I would suggest to any practitioner who, conversant with acute uræmia as it is seen in the human subject—say in the child suddenly struck down by suppression of the renal secretion during an attack of scarlet fever—has not seen uræmia as synthetically presented, to perform the experiment I have named on a young rabbit; he will be astounded at the analogy of the symptoms induced with those which he has seen in the human sufferer from uræmic toxæmia.

After death from injection of urea into the body of an animal, the muscles are found slightly darker than natural; the blood very dark and loosely coagulated; the surface of the serous cavities suffused, and not unfrequently lined with serous exudation. The kidneys are intensely congested and dark; the alimentary canal is sometimes suffused over its mucous surface.

With these synthetical facts so unmistakably presented to us, we need not, I think, go further for a cause of uræmia than to the urea. For although creatinine, one of the substances which we have noticed as a possible cause of the uræmic symptoms, is in truth an ammonia, and may therefore ultimately be found to have a physiological effect analogous to that of urea itself, we have a sufficient cause of the phenomena, independently of a consideration of that organic compound. The same remark applies to creatine.

Accepting, therefore, the urea as the primary toxic agent in uræmia, we are led to ask further, whether the effects demonstrated are due to urea itself, *i. e.* to urea acting unchanged as the poison; or, whether it undergoes decomposition, and the veritably acting poison is a product of that decomposition? Whether, in fact, Frerichs is right or wrong in suggesting that the poison is such a product, in the form of carbonate of ammonia?

The inquiry opens two questions. 1. In the blood, during uræmia, is an excess of ammonia an universal

and necessary fact? 2. Does the exhibition of ammonia, in poisonous doses, produce the symptoms of uræmia?

To the first of these questions Dr. Hammond has given a reply decidedly negative. Confirming by a large number of experiments the observation first made by myself, that ammonia is normally present in blood, he questions altogether the deduction of Frerichs, that the presence of the alkali in uræmic blood is to be set down as the cause of the symptoms. I am bound in the main to agree with Hammond; inasmuch as the evidence afforded by Frerichs is not, in this instance, of a character to substantiate on a satisfactory basis a pathological theory.

It is obvious that Frerichs looked on ammonia as an abnormal constituent of blood, and this constitutes his primary error; but there are other suggestions on which the hypothesis is based which do not hold their ground; for instance, in order to explain the resolution of urea into carbonate of ammonia, he is led to suggest the presence of a ferment, of the existence of which there is really no proof.

Again, as Hammond very acutely argues, the methods by which Frerichs endeavoured to establish the presence of ammonia in blood in uræmic cases were illogical. Not content to search simply for the volatile alkali by merely collecting and examining the vapours given off by blood recently drawn, or exposed to blood heat, Frerichs distilled blood and collected an ammoniacal fluid; while in other experiments he added to the blood caustic potassa, and produced the

evolution of an ammoniacal odour. I follow Hammond in asserting, from direct experiment, that by either of these proceedings a blood containing urea would be made to evolve ammonia, granting even that no ammonia were present in such blood previously.

In like manner, Frerichs seems to me to have erred in taking the fact of evolution of ammonia by the breath as evidence of the correctness of his hypothesis, that in uræmia urea is converted into carbonate of ammonia. For, again, the truth on this point is that the evolution of ammonia by the breath is a normal act. So far is this the case, and in such contrast does the fact stand in relation to uræmia, that during the acute symptoms of the disease the presence of ammonia, before well marked in the breath, is often lost altogether; the acute symptoms themselves being the immediate result of the suppressed respiratory excretion.

Once more, while the fact is undeniable that during uræmic coma ammoniacal excretions are often formed in the alimentary canal, I cannot in any way assure myself that the presence of such ammonia implies an excess of the alkali in the blood. I can advance with Hammond much more safely towards explaining this phenomenon on the supposition that urea, which is a very soluble body, is itself excreted first into the canal, and that the ammoniacal compound is formed there, *i. e.*, in the canal, by the action of the contained matters of the canal on the urea. The supposition here put forward is indeed now almost confirmed

by experiment; for Hammond, having given thirty grains of urea to a full grown fasting dog, killed him fifteen minutes later, and found in the stomach a tenacious mucus exhaling a powerful ammoniacal odour.

But while I thus differ from the eminent Frerichs —and I am sure no one can esteem him more than I even in differing from him—as to the precise toxæmic agent presented in uræmia, I admit that there are occasionally acute uræmic cases, in which, towards their finale, either from decomposition of urea in the blood, or from some change in the destruction of tissue coeval with the formation of urea itself, ammonia in absolute excess is presented in the blood, and even in the respiratory products. These cases present many of the symptomatic characters of modified yellow fever. The skin becomes of a bronzed tinge; the breath is ammoniacal; there is vomiting of fluid of melænic colour; and the blood when drawn remains permanently liquid until treated with lime or baryta: the blood in such cases presents an excess of ammonia. I have seen such an example of disease as is here described; but it is not a fair sample of common uræmia, it is uræmic intoxication complicated with hepatic obstruction.

Discarding, then, the hypothesis that in simple uræmia there is necessarily an excess of ammonia in the blood, I pass to the second question. Does the exhibition of ammonia in large doses produce the symptoms of uræmia?

When ammonia is injected into the blood of animals through the veins in sufficient dose to produce symp-

toms at all, the effects exhibited, very violent so long as they last, terminate almost immediately either in death or in recovery. The great symptom evinced is active violent convulsion, tetanic convulsion. In its intensity, this spasm is different from uræmic convulsive movement, and equally different from the convulsion that may be excited by urea; in its termination also it is different from the paroxysm caused by urea; there is no recurrence, it has for its finish death or convalescence; but in the convulsion from urea and in the convulsion of uræmia there is first convulsion, then respite, and so on, over and over again for hours perhaps, or even days.

The physical characters of the two poisons, in fact, necessarily lead to these differences of action.

Ammonia, a diffusible volatile body, spreads out its influence rapidly through every tissue, and strikes an instant effect. By virtue of the same physical properties, however, it escapes as rapidly and leaves the system free. Urea, on the other side, though a very soluble and easily decomposable body, can escape from the blood by transudation only. Hence, when urea has accumulated in the blood by suppression of its main outlet, or by direct introduction into the blood-current, it is transuded into a variety of parts—into the serous cavities, into the alimentary canal, or into the structures of vascular organs. The blood in this way clears itself for long periods of a portion of the poison; then the poison, reaccumulating faster than it can be thrown off, produces its toxæmic effects in all their intensity, in violent convulsions.

The very act of convulsion hastens excretion; the blood is once more relieved; and the effect ceases, to be renewed whenever the proportion of the poison is sufficient to excite the paroxysm.

We cannot, therefore, on analysing the effects of ammonia or its salts in a large dose, come to any other fair conclusion than that the symptoms produced by the ammonia are not identical with those of common uræmia; there is truly an analogy, but not an identity. On the contrary, between the effects produced by the introduction of a large dose of urea into the system, and the symptoms of common uræmia, there is an absolute identity. But it may be suspected that the presence of carbonate of ammonia in less poisonous doses than those referred to above may be sufficient to produce uræmic symptoms. On this point most fortunately we have good evidence, derived not only from experiment on inferior animals, but from observation on the human subject.

In 1856, Dr. Barker, of Bedford, carried out for me a series of important experiments, bearing on the toxical effects of carbonate of ammonia, when given not in poisonous, but in free and continued doses. In one case, a large and healthy dog having been selected for experiment, Dr. Barker commenced on November 29th, to administer to the animal five grains of carbonate of ammonia daily. The dose was repeated each day until December 3rd; no marked effect following, the dose was given twice in the day. Symptoms were now induced which deserved observation; blood drawn from the ear of the animal

coagulated slowly and imperfectly; the corpuscles were scattered, and entirely modified, some collapsed, and all more or less irregular in shape—oval or many-sided. The animal was also unwell, was thirsty, and inactive; yet he took his food, as he had done throughout, with avidity. On the evening of the 9th he vomited. His breath from the first administration was markedly ammoniacal. It was observed, too, that the animal, although he had eaten heartily in the course of the inquiry, was losing flesh.

In the course of the 10th and 11th days of December, the animal received four five-grain doses of the carbonate. On December 12th, two doses of eight grains each were given. On the morning of December 13th, before any dose was administered, the breath was examined, and found markedly ammoniacal. The blood-corpuscles were much more generally modified; there were none perfect. Some were oval, some many-sided, some star-like. The dark central point was absent in all.

On December 14th, an eight-grain dose was administered in the morning; on December 15th and 16th, the same; on December 17th and 18th, ten grains were given each forenoon. On this latter date, a little blood was extracted, and was watched for twenty minutes, during which time it did not coagulate.

On December 19th, 20th, and 21st, ten-grain doses were given each day. On December 21st, the breath was strongly ammoniacal, as before.

Dr. Barker now gave the animal a respite for seven

days. On December 28th, he recommenced by giving a scruple dose of the carbonate in the morning, and a second dose of the same quantity in the evening. These doses produced vomiting and a staggering gait; but the effects passed off in a few minutes.

On December 29th, one scruple dose was given in the morning. On December 30th, a little blood was drawn, which did not coagulate for nearly an hour. After this observation, another scruple dose was administered. On December 31st, a scruple dose was given in the morning and in the evening.

On January 1, 1857, a scruple dose was given; but none on the 2nd. The breath continued markedly ammoniacal on January 1st, 2nd, and 3rd. On the 3rd, a scruple dose was given; two scruple doses on January 4th, and three scruple doses on the 5th. At this point, the animal became decidedly ill; his appetite fell off, and he was prostrated and drowsy. On January 6th and 7th, two scruple doses were given each day; on January 8th, three similar doses; on the 9th, two; and on the 10th, three. During this time, the symptoms above described became more evident; the breath was more strongly ammoniacal; the blood coagulated slowly and feebly, and the red corpuscles underwent remarkable modifications. They assumed various shapes—stellate, many-sided, and oval. Some were entirely disintegrated. All were free from central opacity. They sometimes aggregated loosely in circular groups, lying out flattened, not surface to surface like coins. They continued long in motion, and had but little

mutual attraction. On January 11th, a dose of half a drachm of the carbonate was given, and proved final. A few minutes afterwards, the animal rolled on his side, became convulsed, with opisthotonos, and died.

In another experiment, conducted by myself, a female guinea-pig was placed in a chamber containing 3,350 cubic inches of space, the air of which was kept constantly charged with ammonia vapour. The symptoms produced were very remarkable, and indicated, perhaps as clearly and succinctly as can be desired, the influence of the long continued effects of the volatile alkali on the animal organism.

The experiment commenced on January 19th, 1857; provision was made for the easy introduction of food into the chamber, and also for renewal of the air in a steady stream. From the 19th to the 21st, the animal lived in the vapour without presenting any signs of distress or danger. The only peculiarity was, that her craving for food increased, and that she devoured ravenously all vegetable matter. On the evening of the 21st she was removed from the chamber. She was warm and lively. A little blood drawn from her nose was rather more dusky than natural; and the corpuscles were irregular in form, some being serrated at the edge, others many-sided. They continued for a long time in motion between the glasses, and their force of aggregation was feeble. The blood coagulated in two minutes.

The animal was again placed in the chamber. The

times of supplying the ammonia were made the same; but the amount of ammonia supplied was increased doubly. At first the vapour excited some irritation of the nostrils, but this effect soon wore off.

On January 23rd, the animal became restless, and her breathing was quick and feeble. She took her food ravenously, and seemed as if she wished to be constantly eating; the instinctive desire for vegetable food was predominant. The blood, examined day by day, showed further modification of the corpuscles. A perfect corpuscle was not to be met with. The blood rather dried on the glass than coagulated; and the fibrous net-work was scantily developed. The experiment was rigidly carried on for several days: the ravenous appetite continued, the animal slept well, and there was little variation in the other signs until January 30th. At this date she became very feeble; she reclined on the side in part, and was for the first time unusually sensitive to sounds. The merest noise, a scratch on the side of the chamber, or any rustling sound, at once startled her and made her uneasy. This day she refused milk, but sought more ravenously after green food. In the evening of this day her sight also must have become disturbed; for when food was put into the chamber, instead of going to it at once in her usual way, she sought about for it, without being able to find it. When it was placed under her nose, she ate cabbage leaf with great eagerness. Left quiet, but watched at a little distance, she was seen to recline a little on the left side, and to remain for several hours in a half comatose

state with the ears involuntarily twitching, the limbs occasionally starting, and the breathing sharp and spasmodic, as in singultus. On removing her from the box, the tongue was found dry, the breathing hurried, and the heart-beat feeble, quick, and irregular. When the hand was placed over the back, all the muscles were felt to be tremulous, and now and then the body was momentarily drawn up by a feeble spasmodic movement.

The blood-corpuscles were now so much modified, that it seemed a marvel how the animal could exist. On the ammonia vapour being removed, however, the serious symptoms quickly passed off. The ammonia was given less frequently, but was occasionally repeated in full dose for two or three days, and always with a repetition of the same train of symptoms. Once I carried on the administration till complete coma was produced, and afterwards exposed the animal freely to air. At the end of two hours the signs of recovery commenced.

On February 28th, the administration was altogether withdrawn, and the animal was allowed to run about as usual. The desire for food, especially for green food, was most extraordinary. In a week she seemed as lively and as well as ever, but the blood-corpuscles did not lose their irregularity for several weeks. It was as curious as instructive to watch the process of repair in those little floating cells. Their restoration was coincident with, if not dependent on, a restoration of the plasticity of the liquor sanguinis. Towards the end of April, but not until then, the

blood assumed its normal attributes. The corpuscles were natural in size, shape, and power of aggregation; the colour of the blood was bright on exposure to air; and the fibrous net-work was well defined.

The extreme effects of the volatile alkali on the human subject are not commonly seen in this day. I think I have once observed toxic symptoms from the administration of carbonate of ammonia, and they certainly were not the symptoms of uræmia. Referring back, however, to the past history of medicine, we are enabled largely to widen our experience on this point; for soon after the carbonate of ammonia came into use as a medicine, its virtues, in a variety of cases extremely unlike in character, were so lauded that it was pushed to an extreme degree, and even dangerous effects resulted from its employment. The justly distinguished Huxham was amongst the first to criticise these proceedings, and to indicate that, as the tendency of the volatile alkaline salts, and indeed of all alkaline bodies, is to the dissolution of the blood-corpuscles and the establishment of fluidity, so in cases where the tendency of the disease is to the production of identical changes, the use of the alkaline salts as the remedies can be but an addition of evil to evil. In corroboration of his judgment in this respect, he quotes various examples of the toxic influence of the alkalies; one of which, as unique in its kind, and as bearing directly on our present studies, I shall quote.

A young gentleman of fortune came under the care of Dr. Huxham, owing to symptoms he had in-

duced on himself by a singular fancy. He had habituated himself to the use of great quantities of the volatile salt, such as ladies use for smelling at, and at length would eat the salt as other people, quoth our author, "eat sugared carraway seeds—*a Δριμυφαγεια*, with a vengeance!"

The result was that he brought on a hectic fever, with severe hæmorrhages from the intestines, nose, and gums; every one of his teeth fell out; he could eat nothing solid; he wasted greatly in his flesh; his muscles became soft and flabby; he broke out all over his body in pustules, which itched intolerably; and his urine was excessively high coloured, turbid, and fœtid. With difficulty he was at length persuaded to leave off his pernicious habit; and under the influence of fine and generous wines, asses' milk, and acidulated juices, he lived for several months, but died ultimately as from marasmus.*

We have thus traced, on direct evidence, the chronic symptoms excited by ammonia on the inferior subject and on the human subject. Comparing these with the symptoms of uræmia, we fail in tracing the identity as signally as we failed to trace an identity between the acute symptoms of ammoniacal poisoning and those of uræmia. Briefly to sum up the facts, the chronic effects produced by the ammoniacal poison are those of typhus or yellow fever, rather than of uræmia; there is no paroxysmal convulsion of muscle, but jactitation; there is hæmor-

* A Dissertation on the Malignant Ulcerous Sore-Throat. By John Huxham, M.D., F.R.S. London: 1757. J. Hinton.

rhagic diathesis, which does not pertain to uræmia; there are the outward manifestations of this diathesis in petechial eruptions, and fluxes of blood, none of which are presented in uræmia; while there is little evidence of prolonged coma, which symptom is the leading symptom of uræmia.

But while a distinction may clearly be drawn between the symptoms of uræmic toxæmia and of ammoniacal toxæmia, I must confess that the fatal shaft driven home from either bow leaves pathological consequences, which it would be hard to separate. Requesting the reader (if to this question his interest in it will be the leader of his industry), first to recast the pathological changes, which have already been described as incident to uræmia, I would ask him next to follow out in comparison the following history of the pathological condition, presented by the animal described at pages 184-87, after its subjection to the effects of carbonate of ammonia to a fatal termination.

The body of the animal, sent to me for examination, was inspected about thirty hours after death. The structures were still quite fresh. There was slight cadaveric rigidity. On reflecting back the chest-muscles, they were found remarkably dark in colour, an appearance which extended through the whole muscular system, and indeed through all the soft parts. The muscles were soft in structure, and easily torn. Fluid blood exuded from the divided parts. The lungs, the liver, and the kidneys were dark, and intensely congested with fluid blood. The

liver and kidneys were soft in structure, and blood exuded from incisions made into them. The bladder was charged with urine, which evolved ammonia freely, and which, subjected to heat and nitric acid, was found to be highly albuminous. The specific gravity of the urine was 1035. The brain was congested with dark fluid blood. The stomach was vascular on its inner surface. Along the alimentary canal, vascular spots were here and there met with. There was no effusion into any of the serous cavities. The lungs were congested, and the bronchial mucous membrane was injected in points, but everywhere lined with secretion. The heart was filled with blood on both sides. The blood was entirely fluid; and evolved ammonia so freely, that by holding over it, at a slight distance, a surface of glass moistened with a little dilute hydrochloric acid, the glass, on drying, was frosted with crystals of chloride of ammonium. The blood on both sides of the heart was dark in colour. Each variety was exposed to the air in a small evaporating cup, at a temperature of 65° Fahr. Thus exposed, the specimens continued fluid for thirty hours, and then commenced to coagulate, eventually coagulating so far as to become modelled to the containing cup, and to admit of being turned out of it as a shaped mass. There was, however, no separation of serum. The blood-corpuscles presented the same modification as were observed during life. They were stellate, many-sided, or collapsed; none were perfect.

From the morbid appearances, then, presented after

death, both by uræmia and by ammonia, we might be induced, if we rested on them alone, to base a suspicion that Frerichs' view is correct. But on further inquiry we are led to put aside even this reading of the phenomena observed. For the fact stands out that the morbid appearances from uræmia, so analogous to those produced by ammonia, are probably dependent on changes occurring late in the act of life, and, to a certain extent, on decompositions occurring after life itself has ceased. We are consequently driven back upon the symptoms as the indices from which to read off the effects of the two poisons, and are enforced to allow the *post mortem* signs to remain in each case as the results of changes induced by poisons, which in character closely approach each other, which in their final effects, when they destroy life, leave behind similar imprints, but which exhibit in the course of their action certain specific differences, dependent on the physical differences of the poisons themselves.

One other point only relating to the respective effects of urea and carbonate of ammonia, in relation to uræmic coma, at present occurs to me as requiring special attention. I have already shown that carbonate of ammonia, acting as a poison on an animal body, produces a condition of blood in which the dissolution of the fibrine is so marked, and the disintegration of the blood-corpuscles is so marked, that hæmatine is set free, the fluidified blood is exuded as blood, and a hæmorrhagic condition is elicited. I have shown again that in uræmia this condition is not set up, but that such effusions as are produced

are simply serous in character. This very marked distinction led me to test the relative effects of carbonate of ammonia and of urea on blood recently drawn; the following short record of the experiences thus obtained includes the facts of this inquiry.

Mixed blood drawn from the neck of an ox was used for the experiment: the natural period of coagulation of two thousand grains of this blood was six minutes.

Two thousand grains of the blood was received into a cup containing four grains of ammonia in one fluid-drachm of water. The blood thus treated, when left at rest, remained permanently fluid, and of port wine consistency. On agitation, fibrine could not be whipped out; nevertheless, on addition of caustic baryta at 60°, a portion of this blood readily coagulated with free evolution of ammonia. Examined microscopically, the corpuscles were found almost entirely dissolved.

At the same time, and from the same animal, two thousand grains of blood were received into a bottle containing four grains of urea in one fluid-drachm of water: this blood coagulated in nine minutes. The clot was unusually dark and jelly-like; the corpuscles underwent very little change, until decomposition commenced.

At the same time, two thousand grains more of the blood were drawn into a cup containing eight grains of urea in a fluid-drachm of water. This blood coagulated within a few seconds of nine minutes; it formed also a dark softish clot, and its corpuscles

underwent no peculiar modification prior to putrefaction.

Into another cup there were received two thousand grains of blood; this cup contained sixteen grains of urea, in solution with one drachm of water. The blood coagulated a few seconds earlier than the specimen which was left to coagulate normally; its corpuscles underwent no modification, and the clot formed, though very dark, was moderately firm.

Lastly, two thousand grains of blood from the same animal were caught in a cup containing thirty-two grains of urea dissolved in a drachm of water. Coagulation occurred in five minutes, the clot being dark but firm, and yielding an abundance of serum. The corpuscles were unaltered so long as the mass remained free from putrefaction.

Thus, in respect to the physical effects of urea on blood, we learn the curious fact, that the salt in its concentrate form has no power to hold the blood fluid; but that reduced or diluted it does hold the blood temporarily fluid, although not to the same extent of fluidity as is so often met with in uræmia. This truth elicited, it was necessary to determine the effects of more dilute solutions of urea on blood. For this purpose I selected the blood of sheep, because the effects are more rapidly determined, and the influence of solvent bodies are more thoroughly brought out: the results are herewith given.

Into a cup capable of holding 2000 grains, four grains of urea were placed, dissolved in four fluid-drachms of distilled water. Into this cup were re-

ceived 1760 grains of blood from the neck of a sheep. Coagulation occurred, as nearly as could be determined, in fifty-five seconds. The clot was rather dark, but became red on exposure to the air; it was not very firm, and serum exuded from it freely. On the upper surface of the blood there was a thin separation of fibrine. The blood-corpuscles were much modified in character, being oval-shaped, rounded, and some even almost rectangular. They were reduced in number.

Into a second cup, containing four drachms of simple distilled water, 1760 grains of blood were received from the same animal. The blood coagulated at the same moment as in the previous specimen. The clot, dark at first, became red on exposure to the air, and gave out serum freely. On its upper surface there was also a thin separation of fibrine. The corpuscles were modified precisely as in the preceding observation. The normal period of coagulation of the blood of this animal was forty seconds. The clot naturally formed was of bright red colour and very firm and uniform, showing no fibrinous separation. The corpuscles were natural.

On the same day four grains of urea, with eight fluid-drachms of distilled water, in which the urea was dissolved, were placed in a cup having a capacity of 2000 grains. Into this cup I received from the neck of another sheep 1520 grains of blood. Coagulation occurred in fifty-six seconds. The clot, at first dark, became red on exposure to the air, yielded at once a large exudation of serum, and was loosely

held together. On its upper surface there was a well-marked soft fibrinous coat. The clot soon became very small, and again of deep dark colour. The corpuscles presented the peculiarities of form and scantiness noticed in the last experiments, but in a more marked degree.

Into another cup containing 480 grains of simple distilled water, were received from the neck of the same animal 1520 grains of blood. Coagulation occurred at the same time to a second, as in the preceding observation. The coagulum presented the same characteristic changes, and the corpuscles did not vary in any single particular.

The blood in this animal coagulated naturally in forty seconds. The clot was red from the first, and firm. The corpuscles were normal.

The temperature of the day on which these experiments were made was high, 70° in the shade.

Comparing these experiments one with the other, and comparing them also with another series of experiments on the addition of water to blood, to which I have already drawn the attention of the profession, the conclusion to which I am brought is, that urea in the blood, in such proportions as we consider extreme, as during uræmia in the human subject, does not, *per se*, produce any extra fluidity of blood; and that when it is largely diluted and added to blood, the results presented are due, exclusively, to the physical effects produced by the water.

Comparing further these experimental truths with the pathological conditions of the blood in uræmia,

we find that there is an identity of representation to an extent that is singularly remarkable.

In uræmia, uncomplicated with acute inflammatory action, the blood shows a tendency to soft gelatinous fibrinous separation; the clot produced is soft and dark, and yields abundance of water; the corpuscles are not broken, but rendered irregular and scanty. The hæmatine consequently is not effused; and, although there may be congestion of soft parts and effusions of serum, there is no true hæmorrhagic exudation.

The effects presented are due to the suppression of urinary water, and not to the urea; which, though active in its way as a poison, is not a blood-solvent.

The comparisons here instituted obtain as a general rule; and when that rule is broken, it is through a modification, either in the course of the diseased manifestation itself, or in changes occurring immediately before or after death. In other words, when in uræmia the blood is found permanently fluid and surcharged with ammonia, the fluidity, dependent on the alkaline constituent, is produced either by some unusual circumstance giving origin to an excess of alkali, or by chemical changes occurring in the urea itself, at or after death, by which its conversion into carbonate of ammonia is brought about.

We have thus, step by step, followed out the cause of uræmic coma. We have determined absolutely that the disease depends on suppression of secretion of the urine; we have analysed this secretion, and examined the various constituents; we have excluded

all producing causes except urea; we have turned to synthesis, and have definitely shown that urea is capable of acting as a narcotic poison, and of producing symptoms identical with those of uræmia absolute. Lastly, by the analytical, as well as by the synthetical process, we have proved that the said symptoms, and the pathology coincident with them, are dependent on urea acting primarily as a poison, and not on its resolution into another poison, viz. carbonate of ammonia.

THE CAUSE OF DEATH IN URÆMIA.

The cause of death in uræmia is primarily to be traced to the accumulation of urea in the system. Against fatal accumulation the body seems to hold a long power of resistance, except in cases such as acute scarlet fever, where the obstruction of the kidney is sudden and complete; for the urea, in chronic cases of kidney-disease, appears to find a moderately free exit by other emunctories than the renal organs, especially by the skin.

For a time, therefore, in cases where there is renal obstruction, the urea may be eliminated with sufficient freedom to save the system from immediate and serious mischief; there may be premonitory symptoms, such as occasional drowsiness, but these symptoms become relieved by a sharp purgation or a free perspiration, upon which the impending danger is removed.

But when once the balance is destroyed, when once there is an excess of urea in the blood, although

at first it be slight, one singular and important fact succeeds; the kidneys, embarrassed by the excessive demand made upon their secreting power, become rapidly congested, and, as a result, rapidly resistant to excretion. Thus, when urea is injected into the body of a healthy animal, and death occurs even within an hour, the kidneys are found so charged with blood, that their structure is distended, and when an incision is made into the structure, fluid dark blood copiously exudes from the divided surface.

This observation, first made by Hammond, I have fully confirmed by repeated experiment. It explains clearly the suddenness of acute uræmic attacks, and their persistency often to the death; for as the resistance to the excretion of urea, by its main channel, increases in proportion to the degree of accumulation, so is the fatal influence of the poison brought out with a steadily increasing force.

The train of evils thus set up, urea accumulating, and the kidneys failing in action, congestion of blood commences in them as from two centres, and extends to the whole venous system, with more or less of intensity. The obstruction put on the venous current passes to the arterial; for, the heart being made to act against an universal blockade, the weakest portion of the capillary system will either give way, or permit a portion of its fluid contents to exude through its coats. The latter alternative is the one most common, and effusions of serum are therefore exceedingly frequent in uræmia; indeed, I doubt whether

a marked attack of the disease ever occurs without effusion. The effect of the exudation differs necessarily in different cases; according to the quantity of fluid thrown out, and the place in which it is thrown out, whether in quantity large or small. Thrown into the peritoneal cavity, the alimentary canal, or the pleural cavities, the effused matter may do no more than create a temporary disturbance; nay, it may, for a time, relieve the system from general embarrassment, by securing a removal from the blood of part of the accumulating poison, as well as by relieving the kidney of some amount of congestion. Thrown into the structure of an organ in the direct line of the circulatory stream, the effects of the effusion are much more serious; for now the congestion becomes more imminent, and the all important organs, the lungs, become partners in the evil, in which the brain soon afterwards takes a share. Death may in this way occur from an universal congestion.

Again, the serous effusion may commence in the brain, in the ventricles, or in the subarachnoidean space; and the pressure induced, obscuring first the consciousness, may extend backwards to the medulla, producing embarrassment of the respiration, and the true serous apoplectic state.

These remarks apply to cases where serous effusion only is presented as the result of a general congestion, commencing in the renal organs and reflected from them; but occasionally in patients in whom there is, with a diseased condition of kidney, an

accompanying diseased state of the vessels of the brain, these vessels, very early in the course of the attack, may give way under the pressure, and true sanguineous apoplexy may become allied with uræmic coma as an immediate sequence. Granting, however, that none of these direct complications of the uræmic disorder occur, death takes place, too readily unfortunately, from a more systematic but not less determinate series of causes, which may be summed up as follows.

Firstly: There is the direct toxical effect of the urea, acting by depression on the muscular and nervous systems. Next; there is a direct effect on the vascular organs tending to congestion of their structure, and to embarrassment of the heart and lungs.

Lastly: The blood undergoes physical modifications which render it incapable of supporting the changes which constitute natural life. These modified states of blood, consisting of increase of water, diminution of red corpuscle, modification in physical construction of the remaining cells, and accumulation in the mass of the blood of a true toxic agent, tend to render that fluid incapable of undergoing normal chemical reconstruction in the pulmonic current. Such blood is incapable, at last, of combining with the oxygen of the air; the arterial blood consequently loses its active calorific life-sustaining properties, and the final result is secured by what may be most properly designated apnœa commencing in the circulation.

In every sense, in fine, urea may be considered as a true narcotic poison, resembling to the finest shade, in its *modus operandi*, the action of those fixed bodies, of which opium is the most fitting type, which, given in such proportions that the system can eliminate them in a moderate period of time, produce merely a passing sleep; but given so as to over-tax the eliminating powers, cause the sleep of death by arresting oxidation.

This last explanation of death is applicable also to many other diseases: to typhoid fever; to snake-bite disease; to cholera; and probably to yellow fever. The patient may breathe freely, but his inspired air is not applied; it is, virtually, as though his lungs were occluded; darkened, congested, comatose, senseless, jactitating or convulsed, he dies taking in air which he cannot apply.

ON THE RELATIONS OF URÆMIA TO FORENSIC MEDICINE.

There are many cases in which symptoms of uræmia may be confounded, by anyone who is not specially observant, with symptoms arising from a narcotic poison, wilfully or accidentally administered. These cases leading, as they almost necessarily must, to a legal, inquiry, may prove extremely embarrassing, if they are not properly investigated. In a case which I have described in the commencement of this essay, a hasty conclusion might easily have led to the conviction of a man, who, though trespassing beyond the bounds of his knowledge, was yet guiltless of any act

of injury to the woman who placed herself under his treatment. And so also in the instance of the letter-carrier, the symptoms, and even the pathology, would have readily misled an incautious man as to the absolute cause of death. It may be well, therefore, to point out tersely the relation which uræmia may hold to cases of poisoning by common poisonous agents.

In so far as symptoms are concerned, uræmic coma and convulsion are closely allied with the coma and convulsion incident to belladonna. I have pointed out the distinctions which apply to the diagnosis between these two forms of disease at page 155.

Uræmic poisoning may also be confounded with the effects produced by opium administered in a fatal dose. The difference in respect to symptoms may be gathered also from a preceding page (156); but here, after absolute death, one other differential point may be brought into play with effect; I mean, of course, the institution of analysis for the detection of opium in the tissues.

Difficulties again might arise with regard to those poisons which produce not merely narcotism, but irritant effects on the alimentary mucous surface. Such poisons as veratria, conium, hellebore, and even colchicum, might be included under this description, and indeed the whole class of narcotico-acrid poisons. As the tests for these poisons, moreover, are doubtful, the difficulty of distinguishing between their action and the action of urea would be, or at all events might be, alarmingly great. The nearest

approach to accuracy in any such doubtful instances will be obtained by a careful comparison between the symptoms and pathology actually presented, and the symptoms and pathology of acute uræmia in its simplest and most absolute type. The absolute symptoms of acute uræmia, and the pathology, have been already supplied, at pp. 152-55, 158-65.

Uræmia might be confounded with the symptoms arising from the cyanides, such as the cyanide of potassium, a substance which is now becoming so common a poison for the suicide. Truly, in most cases where this cyanide is taken, the death is so rapid, and the chemical pathology so marked, that uræmia would scarcely be considered as, by possibility, connected with the event. But occasionally cases occur in which, the dose of cyanide taken being small, a slowly developed class of symptoms are set up which, in many respects, are allied to the symptoms of uræmia. In the *Deutsche Klinik*, No. 13 for 1860, Dr. Huseman records an instance in which a healthy man, twenty-one years old, after taking a gulp of a solution of cyanide of potassium, fell prostrate, but, vomiting spontaneously, was restored from immediately impending death. Afterwards he suffered from giddiness, disturbance of mind, coldness, incapability of rising without falling afterwards, and difficulty of respiration; the mouth of the patient was open, the face dark and cyanotic, the eyes fixed, the pupils dilated, and the action of the heart feeble. Sensibility was decreased over the whole body, but there was no spasm. There was no smell of prussic

acid in the breath. The case terminated favourably by free diaphoresis in about three hours.

These, taken as the symptoms arising from a small dose of the cyanide, present many features not unlike those of uræmia rapidly developed. But there are differences which would make the effects of the cyanide clear; there would seem to be less frequent convulsion, greater coldness of surface, and less determinate coma than in uræmia. Besides these facts, in the matters vomited the presence of hydrocyanic acid would be readily detectable.

After death from poisoning by the cyanide of potassium, the pathological indications presented and the chemical analysis which would be instituted, would alike tend to determine the poison. For the cyanide of potassium produces an universal redness and intense congestion of the alimentary surface of the œsophagus, stomach, and superior part of the small intestine; while the tests for the hydrocyanic acid, if the parts have not undergone long decomposition, testify sufficiently the nature of the destructive agent.

So much for cases in which the medico-legal question to be solved consists in the determination of uræmic poisoning, against the effects of other narcotic and irritant substances; but we have yet to consider, in a forensic point of view, another class of cases in which, during the existence of an uræmic condition actively presenting itself or remaining latent, agents are given as medicines which hasten

the effects of the natural disease, and induce, by this indirect means, a fatal result.

Instances are frequently occurring in which the administration of medicines to meet special symptoms of a simple kind, have led to results which have, by the public and even by the profession itself, been attributed to an injudicious application of the remedies employed. Thus, the administration of opium to check a diarrhœa or to soothe pain, produces now and then, although the dose administered shall have been properly apportioned according to general experience, a fatal coma. We say when such examples occur, that the patient suffers from a peculiar constitution or idiosyncrasy; and we extend again the same explanation to cases in which salivation follows the administration of a moderate, or even small, dose of mercury. But we have seen enough, I think, in our present studies respecting uræmia, to read off the meaning of these idiosyncrasies, and to substitute a reality for a name. We have seen that, whenever the uræmic diathesis is present, then the effects both of opium and of mercury are intensified; and the conclusion I would draw from such information is, that in all cases where death succeeds upon the administration in an ordinary dose of a common narcotic or a mercurial medicine, it should be the business of the expert to inquire into the condition of the kidney; to test for albumen any urine that may have been passed by the patient, or retained in his bladder; to examine even the blood for urea; to examine any effused serous fluid for the same substance; to

compare the general symptoms, and the morbid changes, with the absolute signs and sequelæ of uræmia—and in every particular to endeavour to ascertain the relationships that may exist, between the influence of the exciting agent, *per se*, and the diathesis under which the patient has laboured.

So many examples have occurred within these last few years, in which great obscurity and contradictions have arisen in reference to the poisonous properties of particular and common remedies, that too much stress cannot be laid on the importance of the practical inquiry here pointed out, in all future cases similar or identical in character.

TREATMENT OF URÆMIA.

The treatment of uræmia embraces two distinct heads; the prevention of an acute attack, and the application of direct measures on occasions when acute symptoms have, from any cause, been set up.

The principles of prevention in every case where uræmic coma threatens those who are suffering from chronic renal disease, are few, simple, and effective. The first great element in the treatment consists in securing, as far as is possible, a free eliminative action of the skin. Towards the fulfilment of this object, the skin, over its entire surface, should be kept scrupulously clean, by at least one daily ablution. In such ablution it is not requisite to subject the patient to extremes of cold or of heat, nor to violent rubbings, rinsings, and such like water-bath ceremonials. It is sufficient for all purposes if the patient

will keep every part as clean as he does those parts which are exposed to the light. A pail of cold or tepid water in a common bath is all-sufficient; neither need the washing be a long or formal process. To step into the water ankle deep, with a good glove-sponge to wash the surface freely and carefully, and to dry effectually with a rough towel, these are the requirements, and these alone. The whole proceeding is a matter of ten minutes, and, carried out night and morning, is no great encroachment either on time or labour.

Granting, however, that the ablution can be made once only in the day, it had better be conducted at night than in the morning; for during the day a free accumulation of foreign and excreted matters takes place on the skin, to sleep in which is most unwholesome and injurious. During the night this accumulation is trifling in comparison, but still it is better to remove it also in the morning.

Coincidently with free ablution, active exercise of the body is essential; and of all exercise, that on foot is the best. The exercise, in point of duration, may be prolonged, but it should always be so tempered as not to subject the patient to sudden alternations from heat to cold. Nor should the patient, when he has taken a walk or performed any muscular effort to produce a warmth of the surface, linger about in cold, or chilled, or damp air.

In extreme cases, where the suppression of the renal secretion is such as to demand a greater secreting force for the skin, the hot-air bath may

prove very valuable; nay, the immediate risk of an impending comatose attack may be warded off, I believe, by the diaphoresis which the hot-air bath induces. Enthusiastic advocates of the bath, who teach that it is intended to supplant exercise in all who can afford to pay for it, would urge, and do urge, that the said bath is the only remedy in the class of cases now under discussion. I demur to this extreme doctrine; for I know that, in the majority of cases, the simple influences of ablution and exercise constitute the first preventive agencies, and that, so long as the skin maintains a genial function by such agencies, so long all that can be done evenly, and therefore safely, is achieved. But in saying this, I hold no reserve against the bath, in instances where rapid excretion is required. I am not sure, certainly, by what surface the excretion is, in every case, best effected, whether by the alimentary or the cutaneous surface; but in the majority of instances, perhaps cutaneous elimination is the readiest to be excited, the most pleasant, the least irritating, and as a measure for the discharge of urea, the most effective.

The diet of the patient threatened with uræmia should be of the simplest character. Alcoholics in moderate quantities do no harm, but, as it seems to me, good; for they keep the surface of the skin free, and to the circulation supply a gentle stimulus which is much required. All highly seasoned foods should, nevertheless, be avoided, and an excess of nitrogenous foods equally so. Animal food should not be taken

more than once daily. Fruits in their season are useful, and I know of no kind of fruit which may not be taken in the fresh state; preserved fruits appear to derange the digestion, and to be objectionable.

The amount of fluid taken should be moderate; it need not be greatly or ridiculously reduced, but kept within bounds: the adult man, I mean, should not exceed three pints of fluid daily on the average, for to exceed this is to put a strain on the kidneys, the preservation of which from overwork is all-important. And, there is yet another hint of value in reference to diet,—the food taken should always be received in small quantities at a time. This rule, applicable to men and women in all periods of life, is peculiarly forcible in these cases; for the organs of nutrition and excretion have enough to perform when taxed lightly and often; to overwhelm them with material is to stop their action altogether.

Medicinally, the rule in uræmic cases holds good, "that the least done is soonest mended." It may be of service to give a purgative on occasions when there is constipation, and in the absence of a sweating hot-air bath, it may be essential to administer a brisk cathartic. Indeed, as a matter of experience derived from common empirical observation for centuries past, in early stages of uræmic coma (apoplexy, according to the vernacular), the treatment by free purgation has been the universal and, on the whole, the successful method. But in the selection of a

purgative, great care is necessary. To give, in the cases we are considering, a mercurial purge, is simply and positively to give poison. A saline purgative, therefore, or a simple vegetable pill, such as colocynth, or colocynth with a small proportion of gamboge, is sufficient. In cases where the patient is anæmic and feeble, a chalybeate aperient is often advantageously prescribed, and repeated perhaps daily, if the constipation is obstinate or frequent; such a purgative as the following, for instance, is of great benefit:—*Saccharine carbonate of iron, ten grains; carbonate of magnesia, one scruple; sulphate of magnesia, one drachm.* This may be given two or three times a week, or even more frequently.

While the bowels and skin are thus led to active performance of their duties, the kidneys should be left to do as little labour as is in any degree possible: these facts being always kept uppermost in the mind of the practitioner, that the cessation of the excreting function of the kidney indicates at once a tendency to congestion of the renal organs; that congestion, once commenced, passes rapidly to suppression of function; and that, to relieve renal congestion, not the kidney, but some other emunctory channel, must be freely opened. Let me dwell on this point with special force; for one of the greatest errors common to the inexperienced, is to give diuretics to a badly working kidney—an error as unphilosophical and injurious as it is unpardonable.

I have shown that, in some cases of chronic renal disease, with tendency to suppression of urine and

accumulation of urea, there is often set up, as though it were a natural means of relief, a frequent action of the bowels. The bowels may act two or three times a day, and the amount of matter passed may be large and fluid. This state may continue for years, and, so far from being hurtful, may be, in truth, the saving clause. During the period while this lasts, the urine, moderately scanty, may be altogether free from albumen, and the progress of renal disorganisation be greatly prevented. I need hardly conjoin to this description the advice, that so long as the elimination by the bowels is moderate it should not be checked; and, above all, that it should never be checked by opium, *i. e.* by the administration of one narcotic to check the excretion of another. It is possible, nevertheless, that under some special influence a diarrhœal flux may be set up in an uræmic patient which shall exceed the power of the system, and demand, like an ordinary diarrhœa in a healthy individual, the exercise of some means by which it may be checked. In such instances as are now noticed, the use of opium and of grey powder, alone or combined, being carefully avoided, recourse should be had to one of the simple styptic remedies. Tannin and gallic acid are excellent; if they fail, sulphuric acid is the next remedy to resort to: given in free doses with a little ice-water, as in ten-minim doses of the dilute acid of the *London Pharmacopœia*, the remedy will require but little repetition to produce an astringent effect as thorough as may be desired, for it is the natural tendency of the flux to

stop spontaneously when the kidney resumes its action, and when compensation for suppressed action is no longer demanded.

The rules I have here written refer to cases of chronic renal disorder in adults; but they are not less applicable in a general way to cases in which the uræmic condition is brought on more rapidly, as, for example, in cases of scarlet fever; for, in these instances, to encourage free elimination by the skin and bowels, to avoid opiate or mercurial astringents, and, in a sentence, to relieve the kidneys as far as possible, are the points of practice for the prevention of uræmia.

Pass we from these preventive modes of treatment to the second and more difficult problem. When uræmic coma is fully developed, what shall we do? In the earlier stages, we once again resort to the principle of bringing the skin or the bowels into free play. The application of the hot-air bath, used either in a regularly constructed Turkish bath, or by one or other of the means noticed at pages 103 and 104 of this volume, is undoubtedly the best and most direct measure. The use of heated air being from any cause impracticable, the next best plan is free purgation. I believe that a dose of scammony, with a full dose of colchicum wine, is at once the most active and efficient combination. Whether the colchicum has or has not any specific action, inducing the alimentary canal more determinately to throw off products which should normally find their way out of the body by the urine, I cannot say, for I have

made no experimental inquiries on the subject; but practically it does seem that colchicum exerts an effect in relieving the kidney, which is not exerted by other purgative remedies. Hence, in the cases under consideration, colchicum appears to deserve special consideration.

But when uræmic coma is fully developed, and the patient is unconscious and comatose, the means of producing a derivative action on the skin are not readily applied, for the action must, if it is to be useful, be general and long-continued. It is customary to blister in such emergencies; the act is puerile,—mere waste of time, and trifling with death. It is customary to give at once a brisk purgative: the process is sound in principle, but too commonly ineffective, for not only are there difficulties in administering the remedy, but when the coma is well marked, the bowels lose their excretory action, and do not respond readily even to a purgative, although the patient may swallow it. Are we then left without a remedy? No. I am convinced that when, in a case of acute uræmia, the patient is not excessively fat, nor yet poisoned with mercury, nor extremely emaciated, there is one, and only one, remedy to be adopted, and, "evil be to him who evil thinks," that remedy is none other than a free abstraction of blood.

On physiological grounds venesection, in extreme examples of uræmia, comes forward as a natural and effective remedy; for, as there is a soluble poison in the blood, we secure in blood-letting the readiest

means by which to remove the poison directly. But there is yet another advantage in blood-letting: by it we relieve congestion of the visceral organs, and specially of the kidneys; hence it usually obtains that, after the removal of blood, secretion takes place readily, and a response is offered to diaphoretic and purgative remedies which did not before present itself.

Practically, again, the evidence of the value of blood-letting in uræmia is well borne out. I have shown at page 143, what happy results follow venesection during uræmic coma and convulsion occurring in the young, as sequences of scarlet fever. I have shown, at page 147, the advantage of the same remedy in a case where the uræmic symptoms occurred on the administration of mercury to an aged man who had been long subject to renal obstruction: to these I may add another example which, for special reasons, has always rested on my mind.

An old man, who had suffered from frequent attacks of suppression of urine, was seized, in the year 1847, with symptoms of coma and occasional convulsion. The man was resident at a hamlet of the village of Littlebury, in Essex, and, as he was included in the union district of a practitioner whom I was assisting, I was summoned to take charge of the case. The coma was perfect, the convulsion frequent, and the danger imminent. I then, knowing very little about uræmic coma, but beginning to get impregnated with the notions becoming prevalent respecting the lancet, refused to bleed. I had a

blister placed at the back of the neck; I applied cold to the head, and tried, quite ineffectually, to administer a purgative. Called late at night, I attended the next day, expecting nothing but a fatal termination. On the second morning I received, at the surgery, a message that I need not visit the patient again, as another doctor had been called in. Nevertheless, in passing the house soon afterwards, I did venture to make an inquiry how the old gentleman was progressing, and there I found him, sitting up in bed, quite conscious; and two or three days later I saw him, not sitting up in bed, but leaning on his staff at the door of his cottage, as well as he had been for a period long past.

The history of this man's recovery is remarkable. His friends had formed their opinion that he was suffering from apoplexy, and on that point we were not at variance; but having formed this opinion they jumped at the conclusion that, because there was apoplexy, blood should be drawn. They were, consequently, not satisfied with my treatment; and seeing a neighbouring medical practitioner go by, they summoned his aid. Harassed by no scepticisms about the lancet, my compeer propped the old man in bed, tied up his arm and bled away, and, as he bled, sure enough the cloud of unconsciousness cleared off, the pulse rose, the convulsion subsided, and from that moment the dying man recovered. It may be urged, that the process adopted by my learned brother in this case was not of the highest order of

etiquette, and to say the truth, neither was it of the highest order of science, and I felt both facts thoroughly at the time; but inasmuch as he performed the great feat of prolonging the life of a suffering man, I buried my anger beneath the good that had been done, and took, from the facts, a lesson, never to be forgotten.

In plain truth, when we criticise mercilessly the doings of our predecessors because they bled so frequently, we fall into an error as serious as that which we condemn. Our predecessors, though wanting in our advanced knowledge, were not so devoid of common sense and common feeling as to bleed, and continue to bleed, without seeing any advantages from the practice. They did see advantages: they frequently saw cases, such as we have now discussed, in which results, in these days startling and almost miraculous, were achieved by the abstraction of blood. These results were at once encouraging and misleading,—encouraging in that they promised a remedy universal, on the *à priori* principle, in its application;—misleading in that, the nature of the symptoms being neglected in the considerations suggested by the practice, the practitioner was led to inquire, not whether the case was suited for bleeding at all (the suitability being always admitted), but whether the method of carrying out the bleeding was suited to the case. Hence, there arose endless controversies as to the amount of blood to be taken, the number of venesections, the vein from which the blood should be drawn, and such like, to end at

last, as by an inevitable tendency, in the conception that the remedy, to be useful, could not be carried too far. Thus it was brought into general discredit by the effort to make it do too much.

In these days we have departed from the principle of studying remedies in detail, to study symptoms and pathology in detail; and this departure is good and safe. But it should do more than it has done: it should lead us rationally to apply every means of cure that may seem suited to every given form of disease, and to classify the specific remedies for our diseases as accurately as we tabulate the signs by which the diseases are known, and the organic devastations by which their course is marked.

Returning to uræmia: when the coma is profound and death is imminent, and the diagnosis is sure, the remedy is blood-letting. The remedy should rarely be applied more than once, but then boldly, until in truth the oppression is relieved and the consciousness shows proofs of return. Let there be no local bleeding or half-measure; that is merely taking away the life-element without object, without, for an instant, setting the body at liberty from the poison; but let a vein be opened by a free incision, and let the blood flow, until not the quantity taken but the effect produced determine the cessation of the current. We have had an illustration in point, bearing immediately on this advice. In the case recorded in pages 142-4, a child was relieved at once by one copious disgorgement of blood; but when this same child, some weeks later, was seized in the same manner, and treated

this time by local blood-letting; the treatment was followed, not by relief from the symptoms, but by transition to death; the depressing effect of the slow exudation of blood acting probably with the poison.

But while I thus advocate the use of free venesection in extreme cases of uræmia, it is not rational to hold out the belief that the remedy is certain in every case. We may learn, from experiment, an important fact in reference to the remedial value of blood-letting in uræmia. If a dog, in which the function of *both* kidneys is suppressed, be bled when the uræmic symptoms appear, he will for a time rally from them; he will take food, lose his convulsions, and, in fact, temporarily recover. Again the urea and the water will accumulate; again the symptoms will present themselves, and again they may be relieved by the abstraction of blood. Life can thus be preserved twice, and even three times as long as would be the case were the animal left simply to its fate; but the remedy is not potent to cure, because its frequent repetition is itself a fatal procedure. Thus, in this experiment, the remedy is palliative only. But if another similar animal, in which the function of *one* kidney is arrested, be bled when the symptoms of uræmic coma and convulsion have appeared, they will disappear on the abstraction of blood; if they reappear, the symptoms may once more be relieved by venesection; and now, if the bleeding be effectually done, the relief will often be permanent. The one remaining kidney, freed from its con-

gestion, will recommence its action, and recovery will begin in earnest.

In the case of the human sufferer subjected to the uræmic poison, the effect of abstraction of blood will turn, as in the experimental cases, on the degree of occlusion in the kidney. If both kidneys be so far diseased beyond mere congestion, from structural degeneration or tubular blocking, that secretion is entirely checked, we cannot expect more than temporary relief from abstraction of blood; but in truth such extreme changes are exceptional, and therefore is blood-letting a rational and proper remedy in all cases, selected according to the principles which I have endeavoured now to supply.

Further, in uræmia, one of the great dangers of venesection, frequently encountered in certain forms of active disease, is not to be feared. In inflammatory disorders, for which venesection has been so egregiously extolled, and yet so loudly condemned, the tendency of the blood is to fibrinous deposition, and the tendency of abstraction of blood, unless performed very early indeed, is to the same result. Hence, blood-letting in inflammation destroys not by absolutely emptying the system of blood, as is commonly thought, but by producing a deposit of the fibrinous part of the blood in the heart, which deposit mechanically arrests the circulation. In uræmia, this tendency of the blood to deposition (except there be inflammatory complication) is absent, and therewith the danger, incident in other cases to the removal of blood, is absent likewise.

Finally, when in uræmic coma blood has been drawn and the congestive symptoms have been relieved, and the consciousness has returned, it remains still to relieve the kidneys of extra work, and to save them from congestion, by continuously keeping up a free but safe eliminative action from the cutaneous and alimentary surfaces. If the patient be rendered feeble, he may be supported with good food, with steel, and even with wine; but moderate over-secretion must never be checked, under the fear of the depression it may cause; for now vicarious elimination is a substantial aid to life, and a purgative or diaphoretic, used within reasonable bounds, is virtually a tonic remedy.

ESSAY VII.

ON CARDIAC APNŒA.

By the term cardiac apnœa, I mean a form of breathlessness often terminating in death, which is due to the fact that, in the cases in which it is presented, the pulmonic current of blood is being withdrawn from the air. In this apnœa, the entrance of air into the lungs may be entirely unobstructed; for it is, as its name implies, an apnœa commencing exclusively in the circulation, and at the heart. The symptoms by which this form of breathlessness is characterised, have been noticed at different times by various names: such as, "angina pectoris," "cardiac asthma," "spasmodic respiration," and "syncopal asphyxia." But these terms have only served to connect the symptoms with some particular form of disease, and have been introduced without any specific attempt to define the exact cause of the symptoms as symptoms, *sui generis*.

Further, cardiac apnœa has often been confounded with syncope, from an impression, very generally diffused, that whilst all failing states of the body, depending on arrest of the blood in its circuit, pertain solely to syncope, so all failing states

of the body, depending on an obstruction to the entrance of air into the lungs, pertain solely to dyspnœa and apnœa. These definitions of syncope and of apnœa are imperfect. Syncope, in its pure form, is not attended by any difficulty of the respiration; neither by dyspnœa nor by apnœa. The respiration fails in syncope from want of power in the muscles; but this failure is general in the muscular system, and does not stand out as marked by any special embarrassment of the respiratory mechanism. Apnœa, in its turn, embraces all conditions in which the respiration is embarrassed, and in which the patient feels an inability to respire with natural freedom. Apnœa may, consequently, be divided into several varieties, and designated according to its cause. Thus, if it should arise from obstruction in the larynx, the apnœa would be "laryngeal;" if in the bronchial tubes, "bronchial;" if in the structure of the lung, "pneumonic;" if from arrest of blood to the lungs, "cardiac." For be it observed that the apnœa from arrest of blood on the right side of the heart has, for its essential character, a disturbance between the normal relations of air and blood. The blood inspires and expires; and the production of apnœa is as perfect and as obvious in effect when the blood is cut off from the air, as when the air is cut off from the blood. Such differences as exist in the forms of apnœa are, it is true, definable and objectively striking, but essentially the forms are all alike in effect.

I purpose in the following essay to bring into one

view cardiac apnœa, under whatever circumstances it may arise; to follow it as a distinctive indication into those various forms of disease in which it may develope itself; and to show that, in whatever disease it is recognised, it has one essential cause and a specific importance.

OBSERVED FACTS AND ANALYSES.

A male child, seven years of age, was seized with symptoms indicating laryngeal inflammation. The attack was slight; and, after the application of two leeches to the throat and the administration of antimony for a few hours, the inflammatory signs seemed entirely broken. I left the child on the second evening much better, breathing freely, and playing in its bed. Soon afterwards it fell asleep, but awoke at four on the third morning, uttering a scream. When the parents turned to it, they saw that it was pale as marble. Its face and hands were cold; the efforts at breathing were terrible; and the action of the heart was such that the movements of the bed-clothes indicated every pulsation. I was summoned, and arrived to see these symptoms in all their fatality. I questioned the little sufferer if he were in pain; he pointed to his chest as the seat of the oppression. Constantly restless, he would raise himself on his elbows to fix his chest, and fill it by one long and painful gasp. The stethoscope showed that there was no obstruction anywhere to the entrance of air to the lungs. The first sound of the heart was faint, the second clear; but the action was irregular and

vehement. After a few hours, the chest-wall was raised, and indications of emphysema were presented. At 2 P.M. death closed the struggle. After death I found, as I had suspected, a large fibrinous deposit filling the right auricle of the heart, extending into the ventricle, and so into the pulmonary artery. The deposit had shut off the blood from the lungs, slowly but effectually. The lungs were blanched, emphysematous, and full of air. The windpipe and larynx were sufficiently free: there was no other seat of disease in this child.*

Mr. Duncan McNab, of Epping, has favoured me with the particulars of the following remarkable case of cardiac apnœa.

A labouring man, who had been suddenly seized in the night with a sense of suffocation, came before Mr. McNab. After struggling awhile for breath, he partially recovered, and lived on for weeks, breathing with great difficulty; the only relief he could obtain was by placing himself in one position, viz., by lying with his face downwards and resting on his elbows. In one of his agonising struggles for breath, he died; and after death it was found that a fibrinous band, having its hold in the ventricle, extended into the pulmonary artery. The point of attachment of this band was on the anterior inner surface of the ventricle, so that, when the body was placed with the face downwards, the band was laid parallel with the inner surface of the wall of the vessel, and of-

* See also an analogous case in my work "On Fibrinous Depositions in the Heart", p. 18.

fered little obstruction; but when the body was reclining on the back, the band stretched obliquely across the artery and obstructed the blood-stream. The lungs were of milk-white colour, and bloodless. The man had consulted Mr. McNab about strange sensations in his heart, a year or two before; but his complaints were then regarded as due to profuse smoking and consequent hypochondriasis.

In these two cases there was mechanical obstruction of blood on the right side, due to deposition of fibrine.

In the year 1854 a medical friend, living close to me, summoned me late at night to see a relative, also a medical man, who had been suddenly seized with spasmodic breathing and pallor. I obeyed at once, and found a powerfully built man sitting, or rather partly sitting and partly lying, in an easy chair, with every muscle in his body fixed. His arms were as rigid as in tetanus; his head was drawn back, his face was deadly pale, and the surface of his skin covered with a cold sweat. The agony of expression exhibited by the sufferer was appalling, and his efforts to breathe were desperate. I listened over the chest rapidly, and found that, whenever a little air could be drawn, it found its way readily enough into the lungs. The heart was beating with great irregularity, and with such vermicular action that neither the systolic nor the diastolic sound could be clearly distinguished, but sometimes one faint sound, probably diastolic, was audible. The muscles were so rigid that it was with extreme difficulty we got him to take a stimulant;

but having succeeded, the action of the heart became more decisive, and the attack for this time passed away. When the patient was composed, he told me that he had had several such seizures, but none so severe. When he had his first attack, he said, he thought he must have been seized with tetanus. The pain usually commenced in the neck, seemed to creep down the anterior part of the chest, and then shot, like a lightning flash, through the chest, fixing the muscles immovably; thence it extended down the limbs, first as a tingling tremor, afterwards as a continuous cramp. Micturition sometimes took place involuntarily during such attacks. He had lived freely. He had been an army surgeon, and had seen active service. He was not himself suspicious of being a sufferer from heart-disease: and, like many medical men, he did not like to think of his own case.

On the following morning, I made a careful exploration of his chest. I detected a spot on the left lateral aspect of the chest, where the two sounds of the heart were feebly to be heard, the second most distinctly, and both without murmur. But, turning to the anterior aspect, at all points midway between the base and apex of the heart, there was entire absence of distinct rhythm: here the cardiac action was what I should designate vermicular in character. There were three or four quick, confused contractions, followed by a gap or pause. When he held his breath by an effort, this pause was much prolonged, and the symptoms of oppression were elicited. I diagnosed disease of the right ventricular wall; and to the

friends and advisers of my patient I intimated the hopelessness of his case. In the way of treatment, I ordered tonics, and forbade violent exercise and over-anxiety. It was the fortune of this patient to consult, on the same day, another member of our profession, but his misfortune that he intentionally held back the fact that any previous opinion of his case had been arrived at. The physician consulted was in the midst of morning engagements, and seeing a strong looking man before him, complaining of nothing but an occasional irregularity of pulse, did not examine the chest at all; but, thinking that the irregularity was purely functional, that the spasmodic attacks were due to flatulency, gave an opinion that there was no reason for any change in life, and that the symptoms would all pass away under digitalis and alteratives, which were forthwith prescribed.

The sufferer went home satisfied, and made no provision for danger. A few months later, he attended a near relative in labour, concluded the delivery, retired to another room, and, with a scream, fell in one of his old spasmodic attacks. There was relaxation of the spasm, and recurrence; but this time there was no escape: he died in the midst of one of the seizures.

I saw another illustration of this kind, also in a medical man, who had lived very freely, and had undergone much exposure to the night air. In this case, signs of hepatic enlargement, for a time, drew attention away from the heart, the true seat of the disease. Ultimately the exact nature of the malady

became only too apparent. The cardiac dyspnœa in this case was, however, never so intense as in the case preceding; but the attacks were much more frequent, and sufficiently distressing. I went down to the country one evening to see this gentleman, and slept in his house. In the night I was awakened suddenly, to find him in his dressing-gown by my bedside, imploring me to do something that should enable him to sleep, or to lie for one hour at rest, or to make one deep inspiration.

In this patient, the lungs gave no indication of obstruction: in him, too, by careful auscultation of the left side, the systolic and diastolic sounds could be made out; but on the right aspect all was confusion—vermicular action. I made the same diagnosis here as in the preceding case; and that diagnosis was correct. The patient died in one of the attacks of cardiac apnœa; and the right side of the heart was found, after death, utterly disorganised; the structure in parts translucent, and tearing like wet paper. The walls on the left side were pale, but of ordinary thickness; the valvular machinery was normal; and the lungs were free from disease. The liver had undergone but little morbid change.

In these cases the cardiac apnœa depended on disorganisation of the right muscular wall of the heart.

I attended, in concert with Dr. Willis, in the year 1851, a man, 45 years of age, who in June of that year was suddenly seized with great languor, and inability to move. Twelve years before, he had suf-

fered from rheumatic fever; but had recovered, as he thought, completely. In March, 1851, he had a second attack of rheumatism, for which he would allow no treatment to be adopted, and after which he was never well. On the 16th of June, when I first saw him, he was sitting in a chair, his face presenting extreme anxiety; his gait was feeble and tremulous; his limbs restless; and his mind irritable. On examining the chest, the lungs were found free from any disease. The heart was enormously enlarged, and, as it seemed, pushed downwards, so as to displace the liver; the point of visible pulsation was an inch and a half above the umbilicus, but during a deep inspiration the pulsation was observable immediately over the umbilicus. The action of the heart was irregular, the sounds indistinct. On listening to this heart, it conveyed the idea of a large, soft, India-rubber bag filled with fluid, on which the walls of the bag were irregularly contracting. The pulse was small and irregular; the veins were turgid; and there was regurgative pulsation of the jugulars. The skin was of yellow paleness; but there was no indication of enlargement of the liver, nor of dropsy. The secretions were natural.

The marked subjective feature of this case, again, was cardiac apnœa. But it occurred somewhat otherwise than in the other cases. The man himself was conscious of the want of air, and the anxiety for air during acute paroxysms was as great in his case as in the others; but throughout the respiration did not, to the observer, appear so laboured. Whenever

the patient was asked to take a long inspiration, he could do it, and the transmission of the air into the lungs was, to the ear, freely accomplished. The pain which attended the paroxysms was referred rather to the back and abdomen than to the sternum.

The man continued under our observation without evincing much change of symptom until August, when he was almost altogether prevented from sleep by the recurrence of the attacks whenever he resigned himself to repose. On the 6th of August, during one of the paroxysms of breathlessness, he threw up some blood, bright, red, and frothy. This did not cause an increase of the paroxysms, but rather lightened them. By this time the breathing had become distinctly oppressed; the chest was fixed; and the pain in the back and abdomen during a paroxysm was greatly increased. On the morning of the 10th, he felt slightly relieved; his breathing was free, and he could hold his breath as long as I could hold mine. But the relief was only transient. At nine in the evening, his oppression from want of air, and his efforts to bring the contracted chest into play, were terrible: withal, he was collected. I detected now that there was some effusion taking place in the right pleural cavity. At eleven o'clock, he died while I was present. He rose up in bed, and, saying that another fit was coming on, fixed himself to meet it, with his clenched hands buried in a pillow on each side of him: he opened his mouth widely, as if to inspire, and there he sat fixed as a statue, as I had often seen him before. But this time was the last. I waited,

and found there was no pulsation going on at the wrist; yet the features, drawn firmly in expression of inscrutable agony, continued so fixed, that I did not know whether he was living or not. We put pillows behind him, and gradually he relaxed upon them, unmistakably a dead man.

The diagnosis I had formed in this case was, that there was a source of obstruction to the circulation through the right side of the heart; but I thought that obstruction was in the cavity of the ventricle, consisting, perhaps, of an organised fibrinous concretion. The latter part of the diagnosis was incorrect; for the *post mortem* examination revealed a heart enormously enlarged from dilatation; the pericardium adherent over the whole surface; a firm adhesion between a large surface of the pericardium and the diaphragm; and a girdle of ossific matter, two lines thick and half an inch broad, surrounding the heart almost completely. The bony ring was imperfect only for a short distance over the left ventricle. The right ventricle was large enough to enclose a full sized orange, and its walls were hypertrophied. It had evidently been doing its work as a fixed ventricle.

There was some effusion into the right pleural cavity; but the lungs were normal. The mouths of all the great vessels were much dilated. There were no separated fibrinous concretions; but the enlarged right auricle was filled with red coagulated blood. All the other viscera of the body were healthy.

In this illustrative case, the cardiac apnœa was due

to pericardial adhesion and ossification, by which the right side of the heart was disabled.

On November 26th, 1850, a female child, 10 years old, came under my care for debility. She had suffered from a febrile attack; her friends thought from scarlet fever, as there had been a rash on the skin, and as scarlet fever had been present in a house near by. I ordered the child a saline, and afterwards a tonic; and continued to look in upon her daily until the 26th of August. On the morning of that day she seemed so well that I stated the visit to be the last: she was, when I called, actively at play, and had no complaint except of slight stiffness of the neck, which had been present from the first, but was so trifling that in my mind it passed as a little affectation on the part of the child, rather than as a fact. On the evening of the 26th, I was summoned hastily to this child. She had been well all day, but towards the close of the evening had once or twice coughed. At half-past ten she went to bed with her sister, and got into bed making great merriment. As she was sitting down, she gave a scream, and cried for breath. Her sister turning round, saw her face turn bloodlessly pale, her head being drawn back, and her chest heaving: she gave an alarm, and within six minutes I was by the bedside. The patient was reclining on two pillows, with her head drawn back, and the trunk of her body as rigid as marble. She was conscious; and, when I asked her whether she was in pain, she tried to make an affirmative move-

ment with her head, and pointed to the sternum. I rushed home to fetch some sulphuric ether: when I returned, she was in the same position as I had left her, but dead. She remained rigid for some hours.

I opened the body next day. There was a little thin fluid in the pleural cavities, but no adhesions. The lungs were inflated with air, and were not in the least congested; there was a slight frothy mucus in the larger bronchi, but no obstruction in any part of the respiratory channels. The heart was so contracted, that it had the appearance of a walnut, and it felt to the grasp as hard as a walnut. When it was laid open, its rigid contraction was still evident, but on immersion in warm water it relaxed. Its endocardial surface, prior to the relaxation, was approximated, so that really no cavities existed, so firm was the contraction. There was not a trace of blood in the organ; but the large veins leading into the auricle were full to distension. All other parts of the patient's body were normal except the kidneys, which were slightly marbled in appearance. The whole of the soft tissues were pale, from absence of blood.

In this illustration, the cardiac apnœa was due to tonic spasm of the heart.

In the year 1847, a boy, 12 years of age, residing in Saffron Walden, Essex, was induced by his comrades to take a lesson in smoking tobacco. He smoked two or three pipes, and then, to crown the mischief, put a portion of the weed into his mouth and

commenced to chew. He soon spat a portion of tobacco out, but probably swallowed the remainder. These events, taking place in a very short space of time, were quickly followed by terrible effects. After one or two unavailing attempts to vomit, the body of the boy became suddenly rigid as in tetanus, and the face deadly pale. Alarm being now given, my own assistance and that of Mr. Thos. Brown were sought. I arrived first. By the time of my arrival, the rigidity had relaxed, and in lieu of that the limbs were flaccid, as in profound narcotism from chloroform. The boy, nevertheless, was conscious, although he was unable to articulate. There was no pulse at the wrist; but in the respiration the most striking phenomena were exhibited. Between each act, an interval of several seconds occurred, then a sharp, deep inspiration was made, which seemed to draw up the whole body, and produced the most painful contortions of the face, with tremor in the hands. Not apprised at first as to the cause, I ran my ear over the thorax, under the suspicion that a foreign substance had entered the larynx and was producing obstruction; but the air entered freely, and fully inflated the lungs. After a little time, one of the lad's comrades, more candid than the rest, told me the cause of the attack. Mr. Brown coming in at this juncture, and hearing the history, joined with me in trying to produce vomiting by tickling the fauces. This failing, we quickly surrounded the patient with warm flannels and bottles, and, as soon as it could be got ready, introduced into the stomach, by the œsophagus-tube, a full emetic

dose of sulphate of zinc dissolved in warm water. The stomach fortunately responded freely, and gradually the youth began to recover; but for more than twenty-four hours later there were frequent recurrences of attacks of cardiac apnœa. At first these attacks remitted for two or three minutes, then for five or six minutes, and so on, in gradually increased proportion. As they passed away and the boy became able to converse, his dread of the pain, as each paroxysm approached, was acutely expressed, and, as the spasm appeared, he announced it in a sharp scream. He defined the pain precisely as I have heard it defined by the other patients whose histories have been given—a cutting sensation through the chest, attended with utter inability to breathe until relaxation commenced, and then easily re-excited if the attempt to inspire were rapidly made. The attacks recurred even during sleep; but ultimately they subsided, and a good recovery was the result.

In this case, the cardiac apnœa was dependent on the presence, in the blood, of an alkaloidal poison.

I could multiply largely these illustrations; but the typical cases supplied are all-sufficient for the present history. They present cardiac apnœa as the accompaniment of five pathological states affecting the circulation, viz., obstruction on the right side of the heart; degeneration of the cardiac structure; mechanical embarrassment of the heart from external pressure; spasm of the heart itself; and the influence of a poison. These changes from the

normal life include, I believe, all the states in which cardiac apnœa is induced. They may, however, be modified in detail in various ways. Instead of obstruction in the pulmonary artery from concretion, there may be disease of the vessel and narrowing of its diameter. Instead of external adhesion or ossification of the pericardium, there may be pericardial effusion: and, in addition to the poison of tobacco, a vast number of analogous poisons may be placed on the list, such as strychnia, hydrocyanic acid, ammonia, and chloroform. Further, we may add to the list various animal poisons produced in the body, such as the poison of tetanus, and of the fevers of the typhous class.

DIAGNOSIS OF CARDIAC APNŒA, DIFFERENTIAL AND ABSOLUTE.

Differential Diagnosis. From the other forms of apnœa—the laryngeal, the bronchial, and the pneumonic—cardiac apnœa is differentiated by the following facts.

From laryngeal apnœa, cardiac apnœa differs: (*a*) In that the stethoscope declares the absence of any obstruction in the larynx or tracheal tube, and the capability of free inspiration when the chest-wall itself can be raised to produce inspiration. (*b*) In that there is no deep congestion or blueness of the lips or cheeks; but, in place of these indications, a livid pallor. (*c*) In that the muscular contraction is not a rapidly convulsive act, but of the character of tonic spasm, as in tetanus. (*d*) In that the mind is not necessarily obscured, but on the contrary is, as a

general rule, clear, and acutely alive to all impressions. (*e*) In that the patient is conscious of a terrible oppression of the chest, accompanied usually by a sharp lancinating pain extending from the sternum towards the vertebral column.

From bronchial apnœa, cardiac apnœa is differentiated: (*a*) In that it offers no indication by the stethoscope of bronchial obstruction, no cooing sounds, no *râle*. (*b*) In that there is no convulsive cough, nor expectoration. (*c*) In that there is pallor of the countenance, in lieu of turgescence and dark discoloration. (*d*) In that the inspirations are distant, and as it were withheld, instead of being hurried and short. (*e*) In that the mind is conscious, and vividly alive to pain and anxiety. (*f*) In that the spasm of the general muscular system is tetanic rather than convulsive.

From pneumonic apnœa, cardiac apnœa is distinguished: (*a*) In that there is no pneumonic crepitation, no *râle*, and no dulness of the chest on percussion. (*b*) In that there is no expectoration. (*c*) In that the countenance is of a pallid, instead of being of a bronzed tint, and turgescent. (*d*) In that the mind is conscious, instead of being confused and excited. (*e*) In that the muscular system is tetanic, in lieu of being tremulous or rapidly convulsed.

Lastly, there are two particular symptoms which afford distinguishing marks between all the above named varied forms of apnœa, and cardiac apnœa. 1. The pulse, in other varieties of apnœa, may be slow, or slow and feeble; in cardiac apnœa it is

feeble and intermittent, being often for a long interval absent altogether, and on its return irregular and wiry. 2. In the three more ordinary forms of apnœa, the pulse, if reduced or absent, returns when the difficulty to respiration, whatever it may be, is removed; in other words, *the pulse waits for the respiration.* In cardiac apnœa, the reverse obtains; the respiration is never relieved until there is returning pulse; in other words, *the respiration waits for the heart.*

From *tetanus*, which cardiac apnœa, in its severest forms, closely resembles, it may usually be distinguished by the circumstance that the attacks of apnœa have come on gradually, and with some preceding general derangement of the health, distinct from anything like a traumatic cause. The spasm, moreover, commences in the chest, and does not progress slowly, and as it were muscle by muscle, but is instantaneous, so as immediately to constrict and embarrass the respiration. Further, the spasm does not affect the limbs and muscles of the neck and back so manifestly as in tetanus, while such spasm as is excited is more prolonged in character during a single paroxysm. Lastly, in cardiac apnœa, the symptoms of one attack are usually completed in one paroxysm; this paroxysm may be very prolonged, but, once subsiding, will not recur; while in tetanus there will be alternation of spasm and relaxation.

From *strychnine tetanus* cardiac apnœa is differentiated with more difficulty than from trau-

matic tetanus; or I had perhaps better said that greater difficulties may be presented. Still, there are distinctions in cases where the effects of the alkaloid strychnine are extended over a long period of time. These distinctions consist first in the character of the spasm; the spasm in cardiac apnœa being from the commencement of the attack mainly directed to the chest, while in strychnine tetanus, as in ordinary tetanus, the spasm assails the muscular system more generally, convulsing the body in all its parts. Again, as we have before seen, in cardiac apnœa the paroxysm, however long it may continue, does not recur when it has once subsided; while in strychnine tetanus the paroxysms are repeated in most cases, an entire subsidence of spasm taking place between each. Lastly, in fatal cases there remains, of course, the chemical test, which in cardiac apnœa will necessarily yield negative results; while in strychnine tetanus it will as necessarily yield affirmative evidence of the presence of the specific poison in either the blood, the tissues, or the excretions.

In any given case of poisoning by strychnine, in which the symptoms from their commencement to their fatal termination should consist of one prolonged and unbroken spasmodic representation, it might be next to impossible to determine from the observation of them alone the precise difference between strychnism and cardiac apnœa. In such a case, therefore, the solution of the question involved must be determined by the pathological and chemical investi-

gations instituted after death. If, on such inquiry, the cause of death should have been cardiac apnœa, there will in the majority of cases be found sufficient evidence of disease, either in the walls of the heart, or in the condition of the blood, of the great blood-vessels, or of the coronary vessels, to account for what has occurred; while the absence of strychnine on chemical research will determine the diagnosis. In strychnine poisoning, on the other hand, the detection of the poison, whatever may be the state of the circulatory system, will be sufficient in itself to secure a direct affirmation of the cause of the symptoms, and the nature of the death.

There is only one other disease which might be confounded with cardiac apnœa; namely, *hysteric spasm.* Here, however, the antecedent history of the hysteric paroxysm, the character of the paroxysm itself, its peculiar intermittent type, its connection with partial syncope and unconsciousness, its freedom from intermission of the circulatory pulsation, its occurrence, in short, without evidence of disease of the heart, and its comparative freedom from danger, will usually free the practitioner from any doubt of having mistaken it for its more formidable rival cardiac apnœa.

Absolute Diagnosis. The absolute diagnosis of cardiac apnœa during the paroxysms may be stated thus. It is an apnœa with open air-passages; not panting breathlessness, but suppressed breathing. The struggle for breath is due to spasmodic contraction of the

muscles of respiration, and consequently is marked by no rapid effort of those muscles to overcome a difficulty in the respiratory tract. The apnœa is described by the patient, if he can express himself, as arising from without, as from external pressure, as though his chest were compressed and stiffened. There is darting pain through the chest—cramp. The other parts of the muscular system, if they are involved, are cramped, not convulsed. The mind is usually unaffected. During the paroxysm there is either irregularity or prolonged absence of the pulse; and the same condition, of necessity, is presented by the heart.

The surface of the body is cold and pale; the countenance stormed with anguish, but not incessant; if the spasm permit, there is constant movement on the part of the patient in the effort to obtain relief.

There is always pain of the acute kind more or less marked. The pain is most frequent and most lancinating between two well defined points—the lower part of the sternum, through the chest, towards the last dorsal vertebra: with this pain the breathing is locked up. I believe this pain to depend on spasmodic contraction of the diaphragm.

There is generally more or less of muscular spasm and pain in other parts of the body. In some cases one limb is thus affected, as one arm, which during the whole seizure may be in a state of intense suffering, with more or less of rigidity: in other cases, where the seizures are very severe, this spasmodic

condition may extend throughout the whole of the muscular system, causing a general tetanic constriction.

In prolonged cases of cardiac apnœa occurring in children, as in examples where the symptoms arise from fibrinous deposit on the right side of the heart, there is very frequently emphysema of the lungs progressing to such an extent that the chest-wall is raised anteriorly: in the cases specified, the occurrences named are certain indications of cardiac obstruction in the right cavities, and of apnœa dependent upon that cause.

In all cases of death from cardiac apnœa, the final act is one of persistent muscular contraction; the heart first failing, the muscles of the chest become fixed from tonic spasm; the voluntary muscles follow next; and the whole body, left more or less rigid, may pass into rigor mortis without any intervening relaxation of the muscular organs.

With these symptoms of cardiac apnœa during the manifestation of an acute paroxysm, we must not fail briefly to recall those which are preliminary, in chronic cases, and which may be said to give the idea of a predisposition to an acute attack. One of the earliest symptoms, then, of impending cardiac apnœa, a symptom which is often experienced for years before the disease marks itself out in its determined character, is a sensation of sinking and exhaustion over the region of the heart. This sensation comes on upon very slight exciting causes: such as

deprivation of food, worry, a little overwork, or any excitement. A determinate act of the mind to bear up against this sensation will sometimes temporarily relieve it: a glass of stimulant taken with warm water will always relieve it: a moderate meal will do the same; or lying in the recumbent position. In some persons these exhaustive signs occur at particular times of the day; as at noon, or in the evening, or at the period of going to rest.

For many months, or even for years, the central exhaustion thus depicted may cause no serious inconvenience. The subject of it may become irritable and excitable, or perhaps melancholic, distrustful, and complaining; but those about him attribute these indications to nervousness or mental irritation. At length some telling event corrects this error: it is seen that the patient often becomes struck with faintness and pallor; that he cannot sleep at night; that his faculties are at times unsteady; and that a small amount of exertion produces breathlessness and objective anxiety. Upon these changes there is quickly engrafted an unmistakable acute apnœal paroxysm, and the extreme extent of the disorder is revealed.

The physical diagnosis, in examples such as are now cited, is often, if it be put into practice, exceedingly trustworthy, and capable of shedding a light, too truthful to be happy, over the prognosis of the case. The first fact that strikes the auscultator is an absence of any pulmonic disease of a kind adequate to the production of the general symptoms; for, as a

general rule, the lungs are free from any serious disorganisation or change. Turning to the heart, he notices next, in a vast majority of cases, that there is no murmur, and no evidence of valvular obstruction. In place of indications of valvular mischief, there is, however, a sign which is specially symptomatic. This is irregularity of action. In instances where this irregularity admits of analysis, the hesitation observed is found to occur in the systole. There are a few strokes of perfect rhythm, lub-dŭp—pause; then lub-dŭp-dŭp—pause—dŭp-dŭp; and now again in rhythm, lub-dŭp—pause. The patient himself is not conscious of this irregularity of the stroke as a particular fact; for there is always a column of blood in the arteries waiting to be pushed on by the systole, and hence one hesitation during a fair interval of naturally repeated strokes is not specially felt. In one patient, who was a long time under my care, the systolic sound was sometimes suspended for such a long period, that it seemed to me impossible that some effect of which the sufferer was sensible, should not be felt. But I was wrong: for, on listening, I would arrange that on a stamp of my foot the patient should note his sensations at that moment; and, although I invariably gave the indicated sign at periods when the intermission was longest, yet I never gained any fact of a peculiarity of sensation, nor observed any obvious indication of failure of power on his part.

And yet persons affected with this cardiac irregularity often tell the physician that they feel, at particular times, a symptom which they attribute to irre-

gularity of the heart: this symptom is a creeping, rising sensation, commencing in the cardiac region, and ascending sharply into the pharynx; it occurs often after food, or at the moment of falling to sleep; it is very oppressive, and a source of great alarm to nervous people. It is always present in persons who are predisposed to cardiac apnœa, but it may be present in others; and it does not depend on an intermittent systole of the heart. It is, I believe, a temporary spasmodic twitch, commencing in the diaphragm, and communicated to the œsophagus: or else it is a reflex act from the terminal branches of the vagus to the branches supplying the pharynx. Any way, it is not cardiac; for during its presence the heart may be beating in the most perfect time and tune. I have said that persons predisposed to cardiac apnœa always experience the sensation just delineated; this depends on the circumstance that such person are inevitably sufferers from dyspepsia. The dyspepsia, I believe, always precedes the cardiac malady, and is, indeed, the basis of it, and a continual attendant.

Together with this dyspepsia, there are soon established an irregularity of the cerebral circulation, attended with frequent giddiness, occasional pains in the head, whistling sounds in the ears, and now and then with dimness of sight: these symptoms all contribute to render the person subject to them immeasureably unhappy; and hence, even in early stages, such persons become melancholic and utterly despondent. In two such examples, which came before

my notice, the patients committed suicide; and in a third instance an attempt at suicide, nearly fatal, was made. The cases, in fact, of this kind constitute an almost distinct series of themselves; the subjects of them running the round of the whole Æsculapian fraternity, satisfied with nobody, and responding to no treatment, however rationally devised.

PATHOLOGY OF CARDIAC APNŒA.

In studying the pathology of cardiac apnœa, we are led naturally in all cases to the heart or the great blood-vessels, as the seats of the morbid changes upon which the symptoms depend. In our previous survey, we have seen, incidentally, a variety of morbid phenomena as attendant on cases of the kind named; so that in this place it is unnecessary to do more than recall attention rather more systematically to these morbid conditions.

The most common lesions in the circulatory system by which cardiac apnœa is produced, are thinning and degeneration of the cardiac walls. These changes may present at least three general types. First, a change in which the muscular structure seems to have undergone a general atrophy and softening, without any actual intramuscular fatty deposit. The muscular tissue in these cases may indeed retain, to a large extent, its natural structural elements; but it has undergone waste, and the fibre is consequently thinned and flabby. This form of degeneration commences, I believe, most frequently on the right side of the heart, giving rise in the first stages of change

merely to an irregularity of pulse, and occasional breathlessness; in time the change extends to the left ventricular wall, upon which symptoms of direct failure of circulation occur, and therewith the earlier symptoms of cardiac apnœa are established.

The second description of change in the cardiac structure consists in deposition of fat within the muscle; and, in fact, in that ordinary modification of muscular fibre, which has been so ably described by Dr. Quain and others as fatty degeneration. I believe that, in most of these cases, the first symptom of debility is shewn in the right side, and that the first general symptoms are identical with those which mark simple atrophy. If there be a distinction, it consists in this; that, in the earlier stages of fatty deposit, the affected structure is increased rather than reduced in dimension, and that the physical signs, consequently, give the idea of hypertrophy of the cardiac wall.

The third description of change may be considered as a mixture of the two preceding: it is very common in aged people, and consists of a thinning of the muscular wall, of large deposition of fat externally, so as almost to enclose the heart in fatty matter, and sometimes of partial destruction of the muscular elements of the organ itself.

Together with these changes, there may now and then be met atheromatous degeneration; as atheromatous deposit on the valves, or atheroma of the coronary artery and aorta. Some of the older writers were of opinion that cardiac apnœa (which they, selecting one symptom alone as the type, were wont

to call angina pectoris) was commonly, and indeed almost exclusively, connected with diseased conditions of the coronary arteries. I am inclined, nevertheless, myself to think, that this was an inference based on a limited number of observations; inasmuch as in my own experience, derived from an examination of seven cases of cardiac apnœa in which death actually occurred during the paroxysm, only one case yielded any evidence of disease of the coronary arteries; and in that case an insufficient amount of deposit was present to produce serious obstruction, or materially to affect the cardiac circulation. It may be possible, however, that in some cases the organic mischief may commence in the coronary system; but that which I want to point out, is the fact that the structural change does not *necessarily* depend on a primary lesion in the circulatory system of the heart.

Whenever the heart has undergone to a serious extent any one of the changes in structure which have been given above, the symptoms of cardiac apnœa are manifested by very slight exciting causes; but it not unfrequently happens, that the symptoms of failure of the heart are repeated over and over again without any fatal result, until some new mechanical obstacle comes in the way to obstruct the passage of blood, and to so embarrass the already feeble organ that it cannot recover itself. Amongst the ultimate causes of death in cases of the kind now under consideration, slight filamentous deposits of fibrine around the tendinous cords of the tricuspid or mitral valves are most common. During the stasis

of blood in the ventricles in the course of an attack there is allowed opportunity for coagulation of a portion of the fibrine: as the heart recovers, the filaments of fibrine thus separated interlace themselves with the chordæ tendineæ, the curtains of the valves are brought into approximation, and the results are, complete arrest of the current of blood through the heart, and death.

While yet this sheet has been in hand, I have had a singularly good illustration of the termination of life in the way here described.

A patient of my friend Dr. Marshall, of Norwood, 74 years of age, had suffered for a long time from cardiac apnœa. The paroxysms usually occurred in the night, often preventing sleep, and offering those characteristic symptoms which I have described in previous pages. On the evening of Friday, the 25th of October, this gentleman went to bed dreading somewhat the possibility of a disturbed night, and a return of thoracic spasm. On the following morning, he was found by his attendant sitting up in bed lifeless. He had put on his dressing-gown, and propped himself with pillows, having, as the evidence seemed to show, risen (as was his custom when a paroxysm was coming on), walked the room, and returned to his couch. On making a *post mortem* examination, we found the heart disorganised in the way named in the third division given above. The organ was loaded with fat; the walls were thin and flabby; the right cavities were full of blood, the left empty; there was atheroma of the mitral valve, and also atheroma-

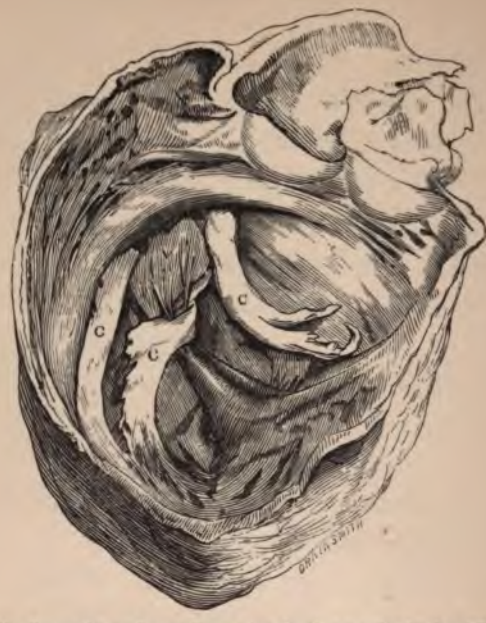


FIG. 1.—C C C. Concretions; Centre Concretion looping up the Cords of Tricuspid Valve.

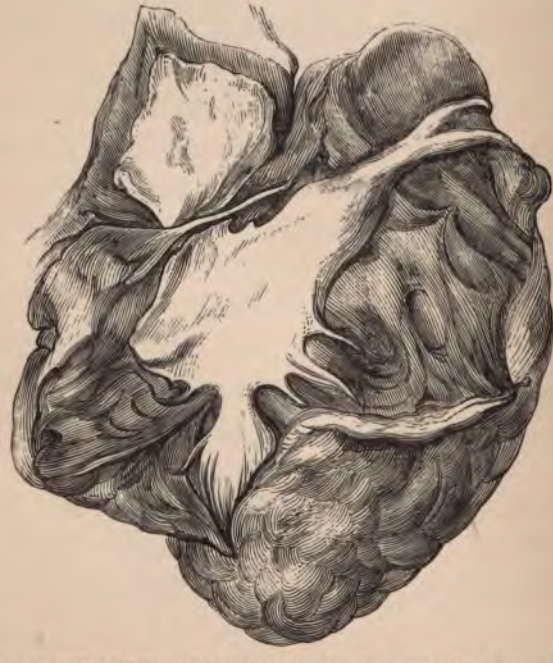
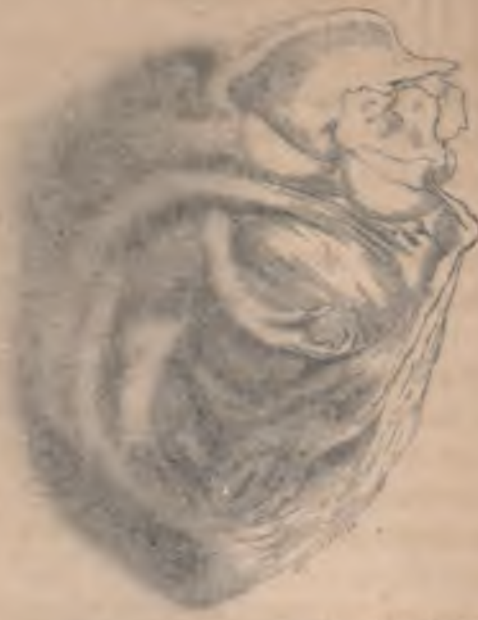
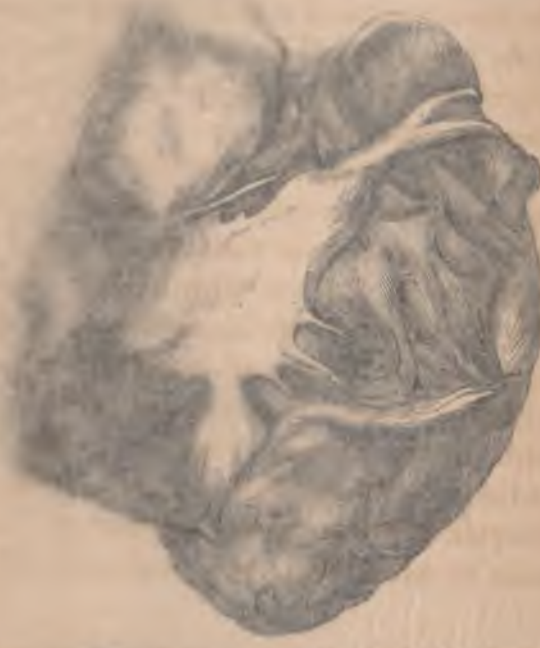


FIG. 2.—Concretion filling Right Auricle and Ventricle, and fixing Tricuspid Valve.



[illegible] Dissection showing up the Cords of Tricuspid Valve.



The [illegible] showing Right Auricle and Ventricle, and Being Tricuspid Valve.

tous deposit along the coronary arteries, but not to such an extent as to reduce their calibre. The final cause of the stoppage of the heart was here found on the right side. The chordæ tendineæ of the tricuspid valve were looped together by filaments of fibrine, so that the curtains of the tricuspid were brought into approximation, and their action suspended. I have described this form of concretion in my work on *Fibrinous Deposition in the Heart*, and have there given a drawing, showing the way in which fibrinous bands may loop up the curtains of the tricuspid, which corresponds so absolutely with the appearances observed in Dr. Marshall's patient, that I reproduce it in fig. 1 of pl. II.

It is necessary, therefore, in examining the heart and blood-vessels in cases of cardiac apnœa, to remember the existence of these filamentous cords, and to look for them with delicate manipulation, as the mechanical obstacles which may have led ultimately to the arrest of the circulation.

The next pathological condition, most frequent after those conditions which have been described, consists in the presence of fibrinous deposition in the cavities of a heart which in other respects is healthy. We find the concretion, giving rise to cardiac apnœa, in a large number of cases of an inflammatory kind; in croup, for example. Here the concretion is usually met with on the right side of the heart, where it may fill the auricle, or the auricle and ventricle, and send a prolongation into the pulmonary artery. Such an extension of concretion is well depicted in fig. 2, pl.

III. Or again, the concretion may take the tubular character, and may produce a cylinder within one of the great vessels, as the pulmonary artery; or it may line a cavity, as the auricle; or it may exist as a solid cylinder grooved on the outside in a spiral form. In the work on fibrinous deposition above cited, I have depicted every one of these varieties of obstruction from deposited fibrine.

In some rare cases, fibrine may be laid down without any inflammatory symptoms; and the indications of cardiac apnœa may occur at intervals, according as the concretion shall lie in regard to the course of the blood; that is to say, according to its position as an obstruction to the blood-current, or its removal from the course of the current. The case, the particulars of which are given at page 227 of this volume, and which occurred in the practice of Mr. McNab, is an apt illustration of the pathological state now being considered.

Again, a concretion may undergo organisation; the organised mass may have a point of attachment, like a pedicle, to some portion of the endocardial surface. It may thus be suspended in the current of the blood; may at times offer no difficulty; but may at other times, under a little extra exertion, float across the current, arrest the blood, and produce a distinct paroxysm of cardiac apnœa. Many years ago, I received the heart of a patient who had been under the care of my friend Mr. Beresford, of Narborough. In the left ventricle of this heart there was a globular concretion, perfectly organised, and at-

tached by a small pedicle to the posterior curtain of the mitral valve.

This patient had for many years suffered from repeated attacks of cardiac breathlessness; in one of these paroxysms she died suddenly, the concretion having been carried upwards into the infundibulum, and having obstructed the arterial stream.

A third pathological condition in cardiac apnœa consists of changes occurring externally to the heart, on its pericardial lining. Thus there may be ossification, as in the case described at pages 231-5; or there may be adhesion; or, again, there may be rapid serous effusion. I remember one remarkable instance of this latter kind. A man was seized suddenly with symptoms of breathlessness, pain shooting through his body, cramps, and extreme coldness. Cholera was dreaded at the time; and, as the patient died without medical aid, it was assumed that he had succumbed to the epidemic. On making the *post mortem* examination, I found the pericardium distended with serum, and no other reason to account for the fatal result; the effusion of course being amply sufficient.

In all the examples given above, there has been such a change from the normal state, that the circulation has become mechanically arrested, either by failure of muscular power, or by internal obstruction to the course of the blood, or by external obstruction to the mechanical action of the heart. I might probably greatly enlarge on these points; I might show other lesions leading to the same result; but the illustrations given are fair types, and those most commonly seen.

There are, nevertheless, a class of cases in which cardiac apnœa exists, and of which the one given at pages 235-6 is an example, where no structural lesion of the heart, external or internal, is present, but where the central organ of the circulation suddenly ceases its function by a spasmodic contraction of its walls. In these instances, the heart is found small, firm, and empty; its cavities virtually obliterated by approximation of their surfaces. This pathological state, uncommonly rare, must have for its cause either a modified condition of blood, by which the tonic contraction is excited, or a modified condition of the nervous system acting upon the organ, to produce spasm through its nervous supply. Up to this time, however, not the slightest clue has been obtained as to the nature of the morbid changes by which the result is brought about.

Lastly, in those forms of cardiac apnœa where the symptoms have been induced by the introduction of a poison from without into the body, we may find the heart, according to the nature of the poison, spasmodically contracted on both sides, or relaxed on both sides and full of blood, or contracted on the left side and relaxed and full of blood on the right. Thus, in poisoning by strychnine, the first of these conditions may be obtained; in poisoning by aconite, the second; and in poisoning by inhalation of chloroform, the third. In these cases the blood, for many hours after death, remains fluid, and generally dark.

Although I have noted the morbid conditions of the heart and blood as holding the place of primary

importance in the pathology of cardiac apnœa, there are one or two points of a general kind which must not be overlooked. As a rule, after the form of death under our consideration the external surface of the body is left blanched and free from ecchymosis. If death shall have taken place in the midst of one of the paroxysms, the body will be left rigid, and this rigidity may pass into rigor mortis without any period of relaxation. But if the death take place after the subsidence of muscular contraction or during a paroxysm in which the muscles of the body generally are not involved, then the body is left free of rigidity, and as though, indeed, it were in a state of gentle sleep. In these cases rigor mortis is slow in its development.

Irrespectively of those lesions which may exist in other organs than the heart, and which may indicate a general degeneration of the body, such as atheroma of the vessels of the brain, fatty degeneration of the liver, and degeneration of the kidneys, but little can be added as specifically denoting, from pathology, the preexistence of cardiac apnœa. But inasmuch as, in a large majority of cases, the fatal paroxysm is due to an arrest of the flow of blood through the *right* side of the heart, so, even in cases where there is no structural lesion of other organs, there is usually found a considerable amount of venous congestion, particularly in the liver, kidneys, spleen, and brain. Even the muscles may share in this congestion, and present a darkened surface, from which fluid blood readily exudes. The condition of the lungs, how-

ever, often differs from that of the other vascular organs in this respect. For, if the circulation be arrested absolutely on the *right* side of the heart, then, the current of blood passing through the pulmonary artery being cut off, the lungs may be left not only free from congestion, but bloodless; while the other vascular organs are largely congested. In cases, on the other hand, where the obstruction has commenced on the *left* side of the heart, and the current of the blood has been cut off in its passage through the aorta, the lungs may be the chief seat of congestion; the other vascular organs being, by contrast, moderately free of blood.

CAUSE AND CAUSATION OF CARDIAC APNŒA.

In considering the cause and causation of the symptoms known in their collected sense under the term cardiac apnœa, we turn in every case to some one or other of the morbid conditions of the heart or blood for a predisposing cause. These conditions may, as we have seen, be varied; but they have all this great feature in common, that they imply a deficiency either in the propelling power of the heart or in the sustaining power of the blood.

One or other of these predisposing causes present, any additional cause may prove the excitant which shall have for its tendency the sudden development of the predisposing cause. Thus, in examples of degenerated heart, mental shock and anxiety, too long a deprivation of food, sudden exertion, passion, sudden fear, diarrhœa, over-repletion—any one of

these—may act as an excitant of the paroxysm. That these excitants differ widely the one from the other is true; but the difference involves no paradox, for a heart enfeebled may be stopped as readily by calling it into a sudden action which it is not prepared to meet, as by withdrawing from it that sustainment of which it is not able to lose a tithe.

But, perhaps, the most frequent of all exciting causes is profound sleep. In the early hours of the morning, when the influence of the sun has been longest from the earth, and when the physical forces have to a considerable extent relaxed under the effects of sleep; when, in simple terms, the action of the heart, even in the healthy man, is at its minimum of propelling power, then are those who are predisposed to cardiac apnœa most readily affected; then not unfrequently they die, perhaps without awaking, or not dying, they awake in the midst of a paroxysm, the chest fixed and the body more or less tetanic. For this same reason, in cases far advanced, the inability to sleep, owing to the recurrence of the paroxysm, becomes, as we have seen, one of the most characteristic manifestations of the affection.

From this it happens that even events, trifling when they occur in the organism of a sound man, may turn the scale fatally in one disposed to cardiac apnœa. For instance, flatulency leading to distension of the stomach, produces in the healthy individual but a temporary inconvenience, a little pain and fulness in waking hours, and at most a disturbed dream or nightmare during sleep. But in a man with a heart

disorganised, when the distended stomach presses on the diaphragm, and inspiratory movements are slightly impeded, and the lung-circulation embarrassed, the feeble heart encounters a struggle for which it has no resources, and an apnœal paroxysm is the result. I believe, indeed, that flatulency, acting in the way suggested, is beyond all others the most frequent exciting cause of cardiac apnœa in cases of cardiac disorganisation; and although, from being a cause temporary in its nature and removable, it is not necessarily a fatal excitant, yet occasionally it is even fatal when the degeneration has reduced the cardiac power to its minimum.

There are again cases, as we have seen, in which cardiac apnœa may occur without any predisposing organic change; but in these instances some poison has been introduced into the blood, and has acted so suddenly and powerfully that the predisposition is not requisite, the exciting cause being immediately brought into action with all-sufficient potency. Poisons which produce these effects act one and all on the same general plan; that is to say, they arrest the action of the heart primarily through the blood. They include a large group of poisons: some of the alkaloids, as strychnine, brucine, and nicotina: the ammonias, the cyanides, and certain of the volatile poisons, such as nitrite of amyl and amylene.

Turning now to the consideration of the symptoms presented during an attack of acute cardiac apnœa, we find them reducible, physiologically, to one leading state—spasm of the muscular system, with its

attendant details of pain and reduction of temperature. The recent progress of physiological science has led us closely to a correct understanding of the reason why this spasm should occur. It has shown us that this spasmodic state need have no organic lesion of the nervous centres for its cause, but that it is a product of a derangement existing between blood and muscle. Let me explain this as tersely as I can.

For the sustainment of the perfect balance of the muscular force, three conditions are absolutely required. First, it is necessary that the muscle should have a full supply of blood. Secondly, it is requisite that the blood thus supplied should possess the power of giving to the muscular structure a permanent temperature, which must not vary materially from 98°. Thirdly, it is essential that the nervous connection betwixt the muscle and nerve-centre be everywhere intact. We can prove these positions by a variety of experiments.

We lay bare the heart of an animal and keep up artificial respiration. If the experiment be neatly done, and the animal operated on be kept gently anæsthetised, the heart may be observed, if the pericardium be unopened, beating rhythmically and with sufficient power to sustain both the pulmonic and systemic circuits for a long period of time. We watch the animal thus placed, and we observe that so long as the circulation is steady there is no convulsion, no spasm. But we interfere with this steady circuit of blood; we compress the great aorta,

for instance, at its origin, and cut off the supply of blood in this way from the systemic circulation; and the results are, first, tremulousness, then convulsion, and finally tonic spasm.

Or, in lieu of mechanically arresting the blood through the aorta, we check it on the venous side, by compressing the superior and inferior cavæ; and now we see the heart cease action and its left side close in permanent systole: upon this the muscles of the systemic circuit become rapidly convulsed, and, as the blood which they retained is applied, spasmodically contracted.

If again, instead of mechanically obstructing the flow of blood, we inject through the pericardium upon the heart a stream of water reduced in temperature nearly to freezing point, we observe the heart make a rigid contraction; whereupon, the central organ ceasing its play and the muscles generally losing their vascular supply, there follow general tremor, convulsion, and spasm.

Once more, we modify the operation by injecting into the heart, through the external jugular, some one of the poisons I have named—ammonia, tobacco, hydrocyanic acid, strychnine; and, as the poison finds its way into the coronary system, we see first in the heart disturbance of action, palpitation, spasmodic contraction, cessation of motion, and afterwards a continuance of these changes carried on to the muscular system at large.

We may vary these experiments in a variety of ways. Instead of laying bare the heart, we may stop

its action by introducing through the thoracic wall a finely pointed pair of forceps, and by compressing the vessels at the base by one firm grip; the results will be the same—general muscular tremor, convulsion, spasm, cessation of motion in spasm.

Or we may reduce the temperature of the blood by making the animal breathe an air intensely chilled; the results will be identical in fact, although the time in which such results will be presented may be considerably lengthened.

Lastly, we may modify this line of research by transferring it from the muscular system at large to some particular muscle. We may inject into the structure of such muscle one or other of the agents to which I have referred, or we may reduce the temperature, or we may cut off its blood-supply; and we shall find as results tremor, convulsion, contraction, more or less persistent.

Between the effects produced by all the agents just described and those effects which are excited on muscle by galvanism, there is moreover a great similarity: there are the same contractions and the same pain; and not only so, but symptoms closely analogous to those of cardiac apnœa may be produced by directing a powerful intermittent shock through the chest from the sternum to the lower division of the vertebral column.

The lessons to be gathered from these observations are very important in regard to cardiac apnœa. They show that, inasmuch as the arrest of blood to a muscle produces spasm, so cardiac apnœa invariably

is excited by any cause that shall rob the heart of the power to feed itself by its coronary vessels, or by any cause that shall so modify the blood as to render it incompetent on entering the coronary system to support the muscle. They show further that the cause of the spasmodic constriction of the chest is the same; that, the heart failing in power, and failing therefore to supply the general muscular system, the involuntary muscles quickly use up their received blood and take on permanent contraction. They explain also why in extreme cases, for the same reasons, the voluntary muscles become similarly implicated.

The question may naturally be asked, why, in all cases of sudden arrest of the heart, the whole voluntary system of muscles is not equally affected with the involuntary during the paroxysm? The answer to this question is exceedingly simple. In all cases of the kind, the arterial system is left charged with blood, and the muscles therefore are, for a certain time, prepared to withstand the arrest. The involuntary muscles, however, go on acting independently and, consequently, use up their blood-supply, and having used it up assume the contractile state; but the voluntary muscles, having no necessity for action, undergo less rapid change and remain longer in a state of relaxation, feeding passively, as it were, on the supply of blood which they had previously received. We see this same fact beautifully illustrated in death from hæmorrhage; as the current of blood is fatally flowing, the heart first ceases, then the muscles of respiration; lastly the voluntary muscles

participate, undergo convulsive movements, and spasmodically contract into inaction.

En résumé, the predisposing cause of cardiac apnœa consists always in gradual failure of the mechanical force of the circulation: the exciting cause is anything that shall drive that failure on to suspension of the circulation, and deprive the muscles of blood. Cardiac apnœa is virtually, therefore, asphyxia commencing in the circulation.

TREATMENT OF CARDIAC APNŒA.

When once cardiac apnœa has proclaimed itself in a definite paroxysm, but little, I fear, can be done to avert a fatal termination during one or other attack. In cases where the paroxysms are slight, or where the warnings only are given, such as have been depicted in the section on "absolute diagnosis," we may, nevertheless, if we are convinced that the change which is progressing consists of degenerative tendency in the structure of the heart, advise the patient very greatly to his advantage, and materially reduce the predisposition. Thus, if he be given to habits of intemperance, by far the most fertile source of cardiac muscular atrophy, we may advise him to reformation, may replace his port wine or brandy by a light Burgundy, and entice him to free himself from that external recklessness, irregularity, and exposure, which, not less than the alcohol itself, lay the foundation of disease. I do not, however, for my own part, in these examples, advise sudden and

complete removal of alcoholics. I believe that to the habitually intemperate alcohol becomes a form of food; and I know that as a food it cannot be instantly replaced by anything else in the way of sustenance, however wisely the new bill of fare may be selected. For this reason, if ever we do attempt rashly to deprive an alcoholic patient of all alcohol, he immediately begins to lose power; his sinking sensations, which no simple food can replace, drive him instinctively from our directions to his old resource; he finds relief in that, and, looking on his Mentor as an impracticable man, returns to alcohol as food, with a more lively appetite for it than ever.

In other cases, where the patient is encumbered with fat, and where it is dreaded that the heart is overburdened externally, or has undergone fatty degeneration, we may render service by recommending such a change in dietary and mode of life as shall tend to reduce the fatty deposit and develope the muscle. In these instances we shall suggest that, as regards food, the patients do reduce the amount of fatty substances, such as butter, or the fat of flesh; next, that they reduce, or remove altogether, substances transformable into fat, especially sugar. I once had under my care a lady, who, encumbered to the last degree with fat deposit, was really in danger. For all articles of diet having a tendency to produce fat, she had a peculiar distaste, except for one, namely sugar; of this she confessed that she would sometimes take half-a-pound, or even a pound a day. I assured her, positively,

that this habit was the cause of her distress, whereupon she had the resolution steadily to give it up; since which time, although she has remained always comely, she has suffered from no serious embarrassment, and has lost all the threatening symptoms of cardiac breathlessness.

Next to reduction of sugar, in these cases, it is well to reduce the amount of beer, and after that of bread; indeed, it is advisable, in most instances, to withdraw the former article altogether, and, if an alcoholic must be taken, to select some of the light French wines, such as claret or Burgundy. In lieu of the fat-forming foods, the patient should be instructed to take the nitrogenous articles of diet in greater quantity, such as eggs, and the lean of meat; while milk, as representing both series of food in perfect proportions, may be used *ad libitum*. Care should also be taken that the meals be frequent, but each time in small quantity, so that the body may never, on the one hand, become depressed from deficient aliment, nor, on the other hand, overburdened by an excessive supply.

To these regulations in diet, exercise out of doors must always be added: this exercise should never be violent, but, commenced in moderate degree, should be steadily increased until the patient is capable of walking ten or fifteen miles a day without undue fatigue.

Together with these rules, free excretion, renal and alvine, should be maintained; and, occasionally, copious exudation from the skin, by means of the

hot air bath, should be excited, if circumstances prevent a due amount of bodily exertion; but I can conceive no case in which the bath can exclude or take the place of muscular exercise.

Medicinally, tonics, and particularly the tincture of the sesquichloride of iron, if there be anæmia, may be given with the greatest benefit. I often prescribe the iron, together with quinine, when there is failure of appetite, and have found the combination excellent; I have also combined it with solution of peroxide of hydrogen, in doses of five minims of the tincture of the sesquichloride, with from one to two drachms of a solution of peroxide containing ten volumes of oxygen, and have seen great good result from this administration. Connected with the use of iron, however, there is one important point of practice always to be borne in mind; namely, to give repeatedly, during the time, gentle alteratives, so as to keep the secretions from the liver and alimentary canal in full play; unless this be done, the tonic acts as an over-repleting agent, and virtually depresses, instead of giving support to the system.

Certain medicines, again, ought always to be carefully avoided; and, beyond all others, mercury. I do not say that a mercurial purgative is to be rigidly prohibited, but anything like a long, or even short course, of mercury, must certainly be avoided. Antimony, too, must, if possible, be struck off the list; and equally so digitalis. I must dwell a moment on this last-named medicine; it happens often in persons predisposed to cardiac apnœa, that the

heart, in embarrassed moments, palpitates—endeavours, in a word, to make up in rapidity what it has lost in power. It is easy, very easy, to be misled by this symptom, and to conclude that an organ apparently so active, but really so feeble, should be depressed into natural action by so direct a sedative as digitalis. The error may almost be called fatal; and, as I have known it do the utmost mischief, I the more heartily expose its dangers.

In those cases where the tendency to cardiac apnœa is induced by deposition of fibrine in the circulatory system, cases of the acute inflammatory kind attended with increase of fibrine, the preventive treatment consists in endeavouring to retain the fibrine in solution; this is best done by the free administration of alkalies. Nay, if a diagnosis can be formed that a fibrinous deposit is left in the heart and is producing chronic symptoms, it would be rational to sustain for long periods of time the superalkaline condition of blood, care being taken not to carry the alkali to the extent of modifying or destroying the red corpuscles.

In other forms of cardiac disease, where there is bony deposit surrounding the heart, or adhesion, or atheroma, or ossification of the coronary artery, I doubt if anything more can be done than to order a well-regulated hygiene, and to protect the patient as far as possible from those exciting causes [illegible] an acute attack is induced.

We have finally to consider the treatr

best during a paroxysm of cardiac apnœa. This is very simple. The first point in practice, is to give the patient plenty of air; the next to place him in the sitting or semi-recumbent position; the third to apply to him as much warmth as can conveniently be borne.

I have shown that the spasm in these cases depends on the removal of blood from muscle; if I had chosen to have gone further into the physiological question, I might have proved that the deprivation of blood from muscle produces spasm, because, under such deprivation, the muscle loses that force upon which its relaxation essentially depends, *caloric.* This is the great law which, remembered, governs the whole of the treatment—*That without a due measure of caloric a muscle must be spasmodically contracted.* It is right, therefore, to infuse warmth by every possible way, by the friction of the warm hand of another person over the contracted chest, by the application of a hot sinapism, by securing for the patient a warm air for inhalation, and by administering fluids heated to a degree such as can be tolerated. It is the customary, I had almost said the instinctive act, of bystanders who witness paroxysms of cardiac apnœa, to give to the sufferer a glass of hot brandy and water or other stimulant; and the immediate success which usually follows this practice is a guarantee of its worth. But it should be remembered that it is not the alcoholic which produces the immediate relaxation, but the diffusible caloric conveyed by the water; and that if the

alcoholic is not at hand, it is good practice to give the heated water without the alcoholic.

From what has been said respecting the administration of caloric during cardiac apnœa, it might be suspected that the warm water bath would be the remedy *par excellence.* There are, however, disadvantages in recommending this: in the first place, it cannot often be obtained in the emergency; in the next place, it does not immediately supply caloric to the internal organs; and in the third place, by largely dilating the vessels of the external surface, it impedes rather than increases the actual force of the circulation. I do not say that in prolonged cases of spasm it should not be tried; but unquestionably the readiest way of supplying caloric to the diaphragm and heart is to throw it into the stomach; there it diffuses quickly, and communicates its relaxing property to all the surrounding muscular organs.

It has been customary in cases of cardiac apnœa to administer what are called antispasmodics, such, for instance, as ammonia, musk, valerian, sulphuric ether, and chloroform. Physiologically, it is entirely false to call these remedies antispasmodics; for, injected into muscle, they have no power of producing relaxation; on the contrary, they are excitants of muscular action, and paralyse by exciting over-action. But there are few examples of cardiac apnœa in which there is not during the paroxysm distension of the stomach from flatus; and in these instances the stimulus, by exciting the stomach to contraction, causes the displacement of the distending air, and

indirectly relieves largely the breathing and the circulation. Hence these substances may be administered with good effect, but they should never be carried so far or repeated so often as to over-stimulate the muscles of respiration or the heart itself.

Direct sedatives, such as opium and hydrocyanic acid, are dangerous remedies during an attack of cardiac apnœa; so, likewise, is alcohol a dangerous remedy if pushed to any extent.

In extremis, artificial respiration is the only resource; the air should be gently introduced into the lungs, and, whenever practicable, should be raised in temperature to a degree equal at least to that of the body itself, 98° Fahr. A simply constructed and portable instrument for quickly warming the air to be used in artificial respiration is a desideratum at the present moment. The simplest apparatus of the kind is one devised by myself and depicted on figure 1 of plate IV. It consists of a small zinc bath, in which are immersed a few yards of fine tubing communicating by one common outlet for admission of air and another common outlet for exit. The opening for the entrance of air in a pair of double-acting bellows (fig. 2, plate IV) is attached by India-rubber tubing to the exit tube of the bath, and the bath itself is then filled with boiling water; in this manner, as the bellows play, the air is drawn through the tubing immersed in the heated water, and is itself very effectually warmed. But the apparatus is cumbersome, and is not, I fear, applicable as yet to the immediate wants of the busy medical practitioner.

FINIS.

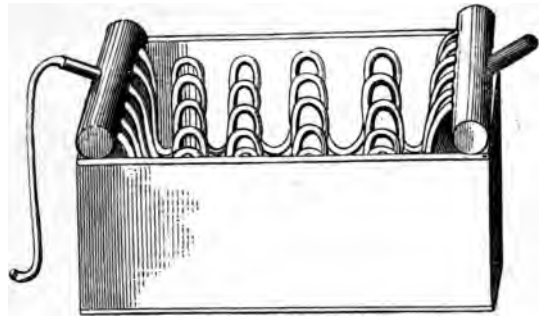


FIG. 1.

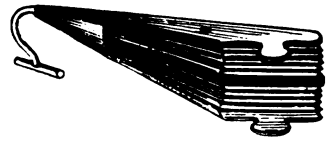


FIG. 2.

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